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MYELOGRAPHY BY THE USE OF PANTOPAQUE IN THE DIAGNOSIS OF HERNIATIONS OF THE INTERVERTEBRAL DISCS*

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INTRODUCTION

NUMEROUS clinical studies have been made during the past ten years concerning lesions of the lumbar intervertebral discs. More recently, interest has been aroused in protrusions of cervical intervertebral discs, largely through the work of Semmes and Murphey[7,8] and Spurling and Scoville.[10] However, the rôle of myelography in the diagnosis of intervertebral disc herniations is still controversial. Many workers in this field have not made use of contrast media routinely because of alleged irritation by the substances employed or of difficulties in performing myelographic examinations and in interpreting findings. Also a hesitancy has been manifest to the injection of a medium which may remain for years in the subarachnoid or epidural space, providing a possible basis for prolongation of symptoms after the original lesion has been removed.

The purpose of this report is to relate our experience with the use of a recently introduced contrast medium, pantopaque. This substance was developed by Strain, Warren and their associates.[11,12] The study is based upon a series of 129 myelographic examinations performed at the Halloran General Hospital. In this group, 77 patients were operated upon and all data available, clinical, roentgenological, surgical and pathological, have been studied. The technique of lumbar and cervical myelography with pantopaque is described and conclusions are presented regarding the significance of myelographic findings and the value of the procedure in the diagnosis of herniations of intervertebral discs. Pantopaque is compared with other agents as a contrast medium for myelography.

TECHNIQUE FOR LUMBAR MYELOGRAPHY

The technique for introduction and removal of pantopaque is substantially the same as that described by Kubik and

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Fig. 1. Patient in position for lumbar or cervical myelography; needle inserted at third lumbar interspace. Note wooden brace attached to fluoroscopic arm to prevent screen from touching needle during roentgenoscopy.

Hampton[4] for lipiodol myelography.

The patient lies prone on a roentgenoscopic tilt-table equipped with a spot film device, his feet braced against the foot rest, a rolled pillow under the abdomen to arch the lumbar spine (Fig. 1). The head of the table is elevated about 10 to 15° above horizontal. Under ordinary aseptic precautions, a No. 18 gauge spinal needle is inserted at the third lumbar interspace, care being taken to make a central puncture. The third lumbar interspace is chosen (even though subsequent removal is slightly more difficult) because of the relative infrequency of herniations at this level. Since most lumbar disc herniations occur at the fourth or fifth interspace, a false defect due to a needle puncture at these levels is avoided. After ascertaining that the needle is within the subarachnoid space, 3 cc. of pantopaque is injected slowly; the obturator is replaced, and the area is covered with a sterile towel. The pillow is removed from beneath the abdomen and placed under the patient's legs.

The fluoroscopic screen is drawn into place and a supporting bar of wood is placed under the arm of the screen to prevent it from dropping and hitting the needle. Bars of several different lengths should be available for use with patients of varying size.

By tilting the table the pantopaque is allowed to flow from the caudal sac to the highest level under study. The pantopaque should be passed over any suspicious area several times. Filling of the nerve root sheaths is facilitated by rolling the patient slightly from side to side. Any lag, delay or deviation in the course of flow of pantopaque should be observed.

As noted by Copleman,[1a] the head of the column should be scrutinized carefully in the anteroposterior and oblique projections, as small central defects may otherwise be overlooked.

If the spinal canal is unusually wide or if doubt exists as to the demonstration of a lesion, an additional 3 cc. of pantopaque may be injected.

The range of motion of the tilt-table is sufficient to allow for study of the lumbosacral canal of the average patient. Occasionally it is necessary to place a pillow under the pelvis in order to tilt the spine sufficiently to fill the upper lumbar canal. The same effect can be produced by having the patient elevate his pelvis several inches from the table, care being taken that the needle does not hit the fluoroscopic screen.

Spot roentgenograms are taken of each lumbar disc. The pantopaque then is pooled opposite any suspicious area and a posteroanterior Bucky roentgenogram is taken on a 10 by 12 inch film (Fig. 6). This

Fig. 2. Pantopaque is pooled in cervical canal. Full extension of neck prevents pantopaque from entering skull.

larger film gives a reverse projection of the canal from that observed roentgenoscopically and offers added evidence regarding the area under study. It is also useful to the surgeon as it outlines the defect in relation to the lower spine. Oblique and lateral projections are of help in occasional cases. The latter are produced by directing the coned rays laterally through the spine with the patient lying prone on the table, the pantopaque pooled opposite the area under study. A wafer grid over the cassette improves detail.

Following completion of study, the pantopaque is pooled about the needle point under roentgenoscopic control and is withdrawn by gentle suction. If a nerve root is drawn against the needle point thus producing pain, rotation of the needle usually will move it away. Occasionally it is necessary to insert a second needle at the fourth or fifth disc level where the pantopaque is withdrawn more readily than at the higher interspaces (Fig. 7).

TECHNIQUE FOR CERVICAL MYELOGRAPHY

The patient lies prone on the tilt-table in the same position as for lumbar myelography. Due to the fact that most roentgenoscopic tables have a limited downward excursion of the head of the table, it is necessary to elevate the patient's pelvis.

Fig. 3. Overhead tube, Bucky and patient in position for roentgenography. The base of the occiput and the inferior border of the mandible are in a plane perpendicular to the table top.

Fig. 4. Bolster has been removed and table tilted nearly to vertical to allow pantopaque to return to lower canal.

This can be done quite effectively by placing a bolster under the patient's abdomen. A simple bolster can be made by rolling two or more pillows tightly together and tying a string or bandage about them.

Six cubic centimeters of pantopaque are injected into the subarachnoid space at the fourth or fifth lumbar interspace, the lower level being preferable to an upper because of the greater ease of subsequent removal.

The patient holds his neck in full extension, thus preventing the pantopaque from entering the cisterna magna (Fig. 2). The fluoroscopic screen is drawn into position, the room darkened and the table tilted so that the pantopaque flows toward the cervical region. As soon as the examiner observes all of the pantopaque in the cervical canal, the table is tilted back to horizontal position. Since in this position the cervical canal is at a slightly lower level than the thoracic canal and the cisterna magna, the pantopaque remains in this position. The patient may then drop his chin slightly so that the base of the occiput and inferior border of the mandible are superimposed on the roentgenoscopic image, thus allowing for more satisfactory visualization of the cervical canal. Slight tilting of the table upward and downward will distribute the pantopaque evenly through the canal.

In this area the pantopaque is spread more thinly than in the lumbar region.

Fig. 5. Normal lumbar myelogram (spot roentgenogram) following injection of 3 cc. of pantopaque through needle at third lumbar interspace. Outline of upper canal as copied from other roentgenograms of the same patient appears above the column of pantopaque.

Consequently the shadow is less homogeneous. However, the nerve root sheaths are demonstrated clearly and defects, when present, are fairly obvious. As in lumbar myelography, it is advisable to allow the pantopaque to flow over the suspected area several times to be certain that any defect noted is constant and represents an actual fixed deformity.

Occasionally the pantopaque will break up into small globules which fail to coalesce. This is less likely to occur if the medium flows smoothly and evenly over the dorsal curve. Small root defects may not be detected if so-called "globulation" is present but large defects are not obscured.

In this region posteroanterior Bucky roentgenograms are superior to spot roentgenograms (Fig. 40). Use of a small focal spot is desirable and a rotating anode tube will give especially fine detail (Fig. 3).

Since most cervical disc lesions involve the sheaths of the sixth or seventh cervical roots which emerge above the corresponding cervical vertebrae, it is rarely necessary to visualize the upper canal. However, this may be done if desired by projecting this portion of the cervical canal through the open mouth, using a technique similar to to that commonly employed for demonstration of the odontoid process.

Following completion of study, the bolster is removed from beneath the patient's abdomen and the table is tilted to a nearly vertical position (Fig. 4), thus causing the pantopaque to return to the lower canal. The skull and upper spine should be examined carefully with the roentgenoscope to be certain that no droplets are present in these areas. The pantopaque is then pooled under the needle point and removed.

Fig. 6. Ten by twelve inch posteroanterior roentgenogram outlines the column of pantopaque with its relations to the lumbosacral spine.

TECHNIQUE FOR THORACIC MYELOGRAPHY

While posterior protrusions of intervertebral discs are infrequent in the thoracic region, this area is occasionally studied myelographically for evidence of other lesions. A technique similar to that employed for cervical myelography is satisfactory for most cases. Occasionally it is advisable to use 9 cc. of pantopaque and to withdraw the needle so that the patient may be roentgenoscoped in the supine position. In such cases, after all studies have been completed, a needle is reinserted at the fourth or fifth lumbar interspace and the pantopaque is withdrawn.

ANALYSIS OF MATERIAL

Myelography was employed as a diagnostic procedure in 129 patients, pantopaque being the contrast medium used in 118, lipiodol in 11. In this group, 77 patients were operated upon.

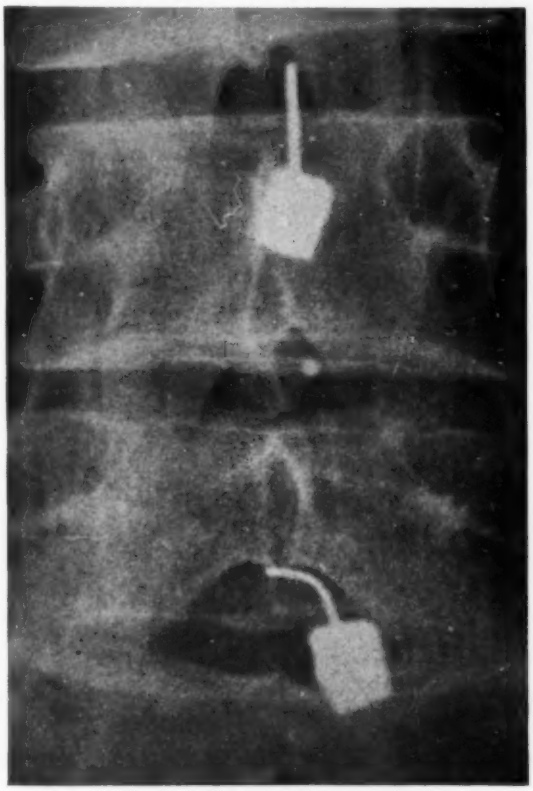

FIG. 7. Complete removal having been impossible by aspiration at the third interspace, a second needle was introduced at the fifth interspace. One small drop of pantopaque remains.

FIG. 8. Case J. T. The right fifth lumbar root defect was overlooked prior to operation. A lateral protrusion was found.

In the cases with positive myelograms, the nature of the lesion was predicted in all but two. In one case (Fig. 13) a herniated left lumbosacral disc was diagnosed whereas at operation an extradural metastatic tumor was found. In the other case, a cervical cord tumor was suggested by the myelogram (Fig. 14) and also by clinical studies; at operation, a herniation of the intervetrebral disc was found between the sixth and seventh cervical vertebrae on the left side with extensive arachnoidal adhesions.

In 64 of the 77 patients the site or sites of lesions were predicted correctly by melographic examination; in each of 8 additional cases, the major herniation was identified by myelography but additional

Fig. 9. Case E. L. Narrow canal with under-developed nerve root sheaths.

slight defects were noted myelographically which were not found at operation to be due to protrusions. In some of these cases the secondary defects were apparently due to arachnoidal adhesions or hypertrophy of the ligamentum flavum; in others, the cause of the defects could not be determined.

Of the 5 remaining cases, the myelogram in one was reported as normal; subsequent review of the myelograms showed a definite minimal defect of the nerve root sheath at the site of the lesion (Fig. 8). One patient had an unusually narrow canal with poorly developed nerve root sheaths (Fig. 9 and 10); although no defect was visible in the opaque column, a moderately large protrusion was found compressing the left fifth lumbar nerve root distal to the arachnoidal extension of the sheath. Each of the other 3 patients had apparent high terminations of the caudal sac, at or above the level of the fifth lumbar disc without perceptible defects in the opaque column or nerve root sheaths. In each of these 3 cases a large protrusion of the fifth disc was found (Fig. 11 and 12.)

TYPES OF DEFECTS

1. "Block" defect from complete obstruction usually produced by a large midline protrusion. The only cases observed in this series were the 3 patients noted above with apparently high terminations of the caudal sac from midline protrusions of the fifth lumbar disc. A roentgenogram of a patient studied by one of us (S. W. G.) in 1937 demonstrates a complete block opposite the second lumbar disc (Fig. 15). Clinically, it is difficult and often impossible to differentiate such lesions from spinal cord tumors. Complete blocks except at the level of the fifth lumbar disc are uncommon; in a

Fig. 10. Same case as in Figure 9. At operation, a large protrusion was found compressing the left fifth lumbar nerve root.

series of more than 200 cases observed by one of us (S. W. G.) and a series of 75 cases studied by another of us (A. B. S.), only 2 such cases were encountered. With a complete block none of the contrast medium passes the site of obstruction, but if pantopaque or lipiodol is introduced above and below the area of block the limits of the obstructing lesion can be determined.

2. "Gap" defect from incomplete obstruction, also usually produced by a large midline protrusion. In this type the pantopaque forms two pools, one above and one below the lesion. On roentgenoscopy the contrast medium is seem to pass slowly over the lesion, drop by drop, usually lateral to the midline. "Gap" defects were present in 4 cases (Fig. 16, 17, 18 and 19); in each, a large herniated disc was found and removed at operation.

3. "Veil" defect. In this type, also caused by a midline herniation, the contrast medium is of decreased density at the site of the lesion due to thinning of the opaque column opposite the protrusion. Myelograms of this type were found in 4 cases, of which Figure 20 is a typical example.

Fig. 11. Case V. G. Spot roentgenogram with patient standing demonstrates apparent high termination of the caudal sac.

Fig. 12. Same case as in Figure 11, lateral roentgenogram. At operation a complete herniation of the fifth lumbar disc was found.

4. "Hour-glass" defect usually due to a bilateral herniation of a disc. This type of defect requires careful scrutiny. If there is a symmetrical defect between two pairs of normal roots or axillary pouches, it is probable that the constriction is due to a slight bulge of the disc or to osteophytes about the margins of the vertebral bodies and not to an actual herniation. False defects of the "hour-glass" type are also apt to be multiple. In a true "hour-glass" defect due to a herniation of an intervertebral disc, there is fusiform constriction of the canal with shortening, elevation, distortion or obliteration of the nerve root sheath or sheaths at the level of the herniation. Defects of this type were found in 5 cases in which exploration confirmed the myelo-

Fig. 13. Case D. S. The lateral fourth lumbar defect was interpreted as a disc protrusion. An epidural tumor was found at operation.

Fig. 14. Case F. F. An extramedullary tumor was suspected opposite the sixth and seventh cervical vertebrae; a large cervical disc herniation was found between these two vertebrae associated with extensive arachnoidal adhesions.

graphic diagnosis (Fig. 21, 22, 23, 24 and 25).

5. "Lateral pressure" defect from lateral herniation of a disc. This is the type encountered most frequently. The appearance is that of a smooth, rounded defect or notch on one side of the column. These defects

Fig. 15. Complete block opposite the second lumbar disc demonstrated with lipiodol (case not in this series).

vary in size but are always accompanied by shortening, amputation or distortion of the nerve root sheath on the side of the lesion. Twenty-one large lateral defects and twenty-four small lateral defects were present in this series. A defect was considered large

Fig. 17. Case F. J. "Gap" defect from large midline protrusion.

if it was at least one-third of the width of the column. Figure 26 is an example of a huge lateral defect. Figures 27 and 28 illustrate other large defects and Figures 29 and 30 demonstrate small lateral defects.

Fig. 16. Case B. G. "Gap" defect produced by large right fifth lumbar disc protrusion. During roentgenoscopy, pantopaque was seen dripping from one pool to the other.

It should be noted that the size of the defect in a myelogram is not always an index of the size of the protrusion. A large defect indicates a large herniation. On the other hand, a small defect does not necessarily mean that a lesion is small, especially at the lumbosacral level or if the canal is narrow. Under these circumstances, most of the protrusion may lie under the root in its extradural course after it leaves the dural sac. Figure 29 demonstrates a very small lateral left fifth lumbar defect with a shortened nerve root sheath; on exploration a large lateral herniation was found.

Fig. 18. Case J. K. "Gap" defect.

Fig. 19. Case P. D. "Gap" defect.

6. "Root" defect from lateral herniation of a disc, compressing the nerve root sheath without obtruding upon the subarachnoid space of the spinal canal. Abnormalities in the filling of the nerve root sheaths not associated with any other abnormalities in the column were found in 21 cases. In 10 cases there was complete failure of filling or "amputation" of the nerve root sheath. In 11 cases the nerve root sheath on the side of the lesion was shorter than the one on the opposite side. The roentgenogram reproduced in Figure 31 is a typical example of amputation of a nerve root sheath on the side of a herniated disc. In Figure 32,

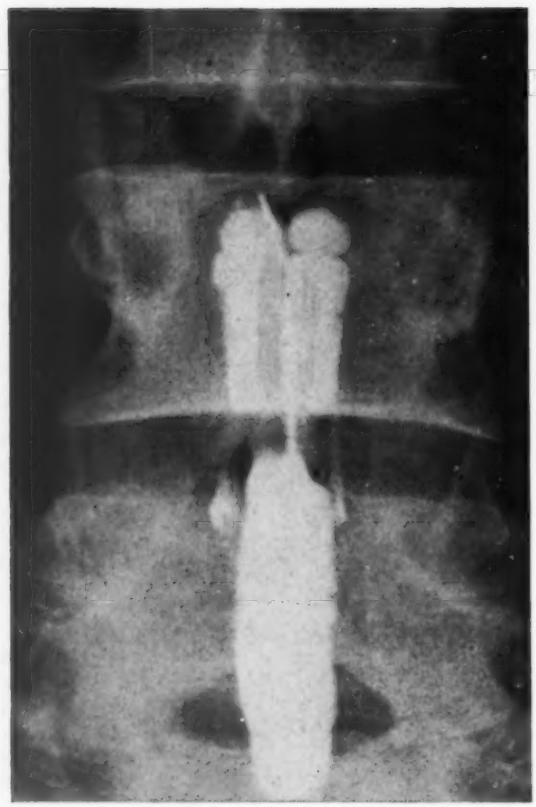

Fig. 20. Case D. S. "Veil" defect from midline protrusion of fourth lumbar disc.

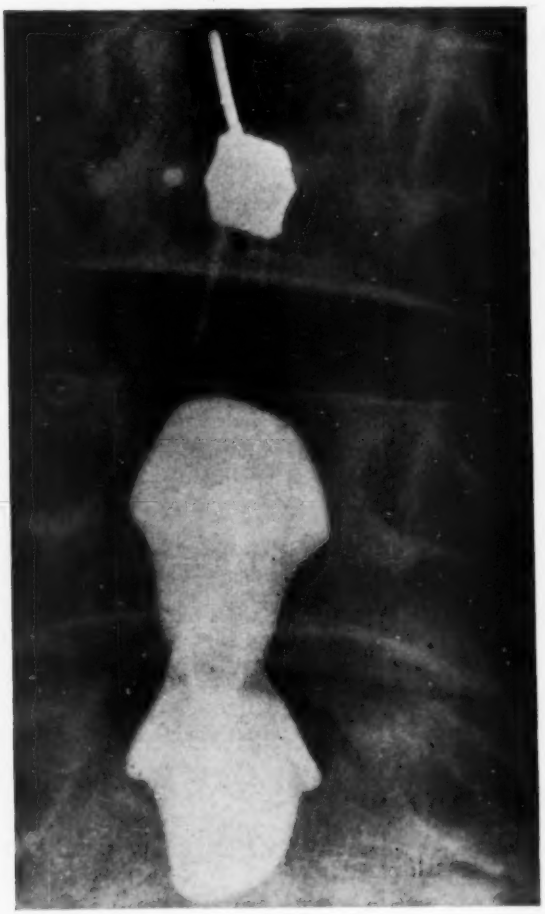

Fig. 21. Case J. R. "Hour-glass" defect from large protrusion which distorted bases of the nerve root sheaths.

the nerve root sheath was shortened and elevated on the side of the lesion while the one on the opposite side was of normal appearance.

MULTIPLE HERNIATIONS OF LUMBAR INTERVERTEBRAL DISCS

In 11 patients two herniated lumbar intervertebral discs were found at operation; in every case the correct diagnosis was predicted from the myelogram. In 6 cases, both herniations were on the same side at the fourth and fifth lumbar discs. In 2 cases, the herniations were on the same side at the third and fourth lumbar discs. In 2 cases, the herniations were in the midline at the fourth lumbar disc and on the left side at the fifth lumbar disc. In one case, the herniation was on the right at the fourth lumbar disc while at the fifth lumbar disc it was on the left (Fig. 33). In this patient, an Italian Service Unit Trainee, the clinical signs pointed to the left fifth lumbar disc as the site of the lesion. Both discs were ex-

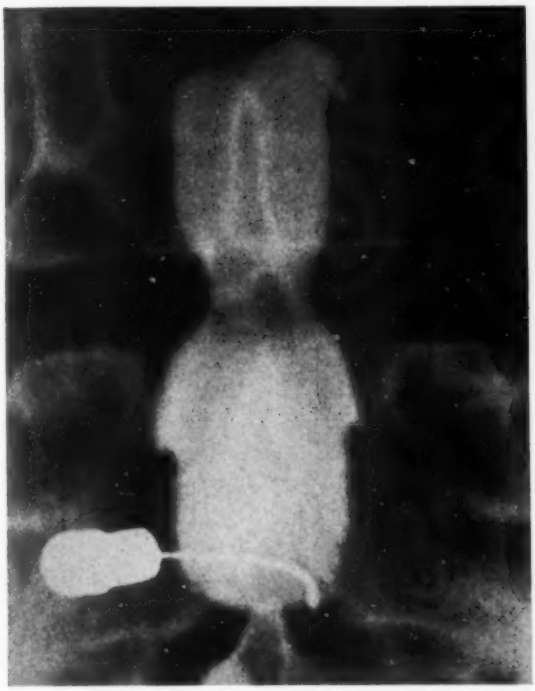

Fig. 22. Case R. H. "Hour-glass" defect; midline herniation was found at operation.

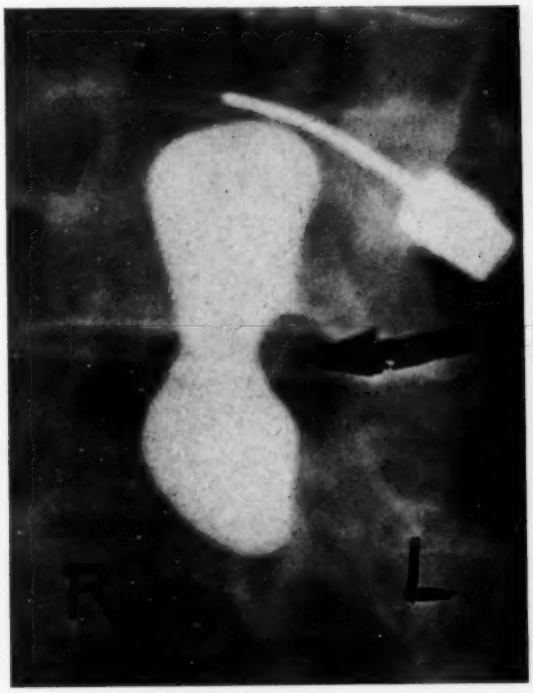

Fig. 23. Case J. M. "Hour-glass" defect from midline herniation.

Fig. 24. Case D. P. Nearly complete "hour-glass" defect.

Fig. 25. Case J. E. "Hour-glass" defect.

plored and large herniations found at both levels. A large tear was found in the annulus of the fourth lumbar disc with extrusion of disc material into the spinal canal.

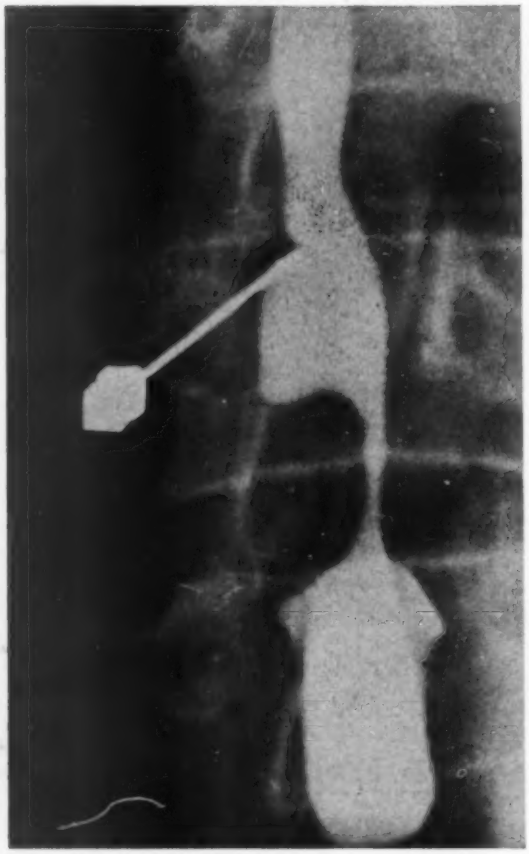

Fig. 26. Case G. M. Huge "lateral pressure" defect; a large fourth lumbar protrusion was found free in the canal.

Fig. 27. Case F. K. "Lateral pressure" defect.

Other examples of double disc herniations are illustrated in Figures 34, 35 and 36.

RECURRENT HERNIATIONS

Myelography was carried out in 3 patients who continued to complain of pain months after herniated discs had been removed. In 2 cases who had had their orig-

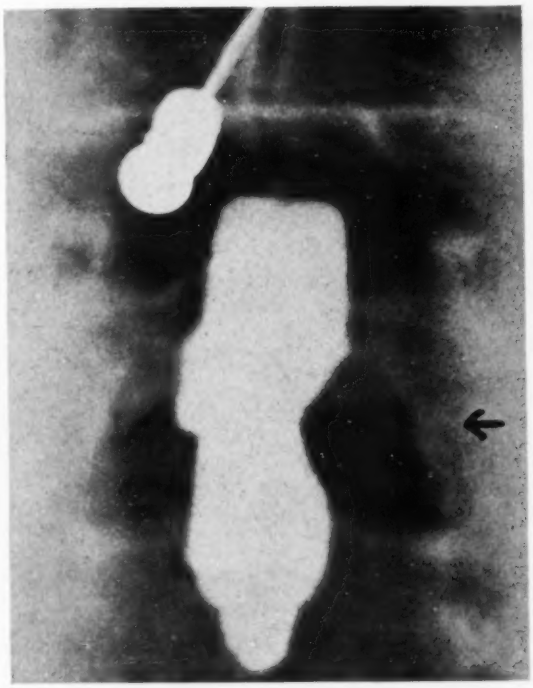

Fig. 28. Case P. C. Left lateral fifth lumbar protrusion producing "lateral pressure" defect.

Fig. 29. Case R. F. Small "lateral pressure" defect, fifth lumbar, left; a large lateral herniation was found at operation.

Fig. 30. Case R. R. Small "lateral pressure" defect, fourth lumbar, right.

Fig. 31. Case N. P. "Amputation" of nerve root sheath by lateral protrusion of fifth lumbar disc on the right.

Fig. 32. Case M. L. Elevation of the left fourth lumbar nerve root sheath; lateral protrusion was found at operation.

Fig. 33. Case E. T. Double protrusions, fourth lumbar right and fifth lumbar left.

inal operations in other institutions, sizable recurrences were demonstrated myelographically and found subsequently at operation (Fig. 37). In the third case a single large fourth lumbar protrusion was found and removed at this hospital; some weeks later myelography demonstrated a large defect at the same level and a caudal sac terminating at the level of the fifth disc. Re-operation disclosed disc protrusions at both levels.

Fig. 34. Case S. G. Double protrusions, fourth lumbar midline and fifth lumbar left.

Postoperative myelographic studies have been made of only 4 other patients, all of whom were symptom free at the time of examination. All showed slight but definite distortions of the canal at the levels of former protrusions but less prominent deformities of the nerve root sheaths. While this

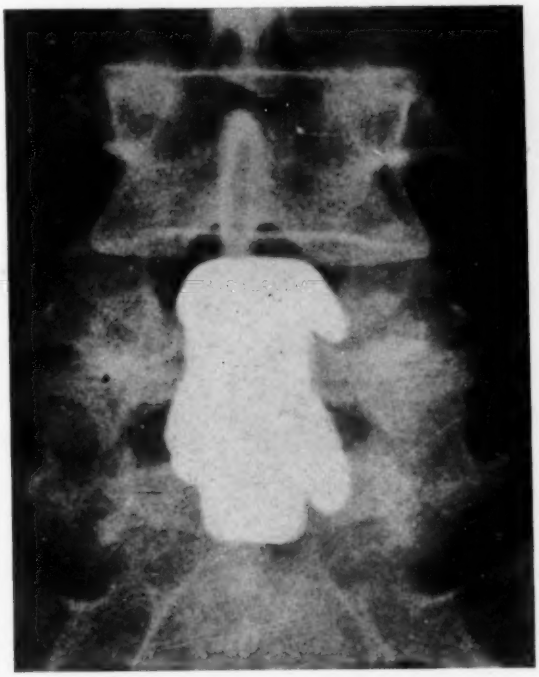

Fig. 36. Case J. R. Elevation of right fourth and fifth lumbar nerve root sheaths; lateral protrusions were found.

series of postoperative studies is small, it is our impression that myelographic findings in this group are not significant unless the defects are large and well defined.

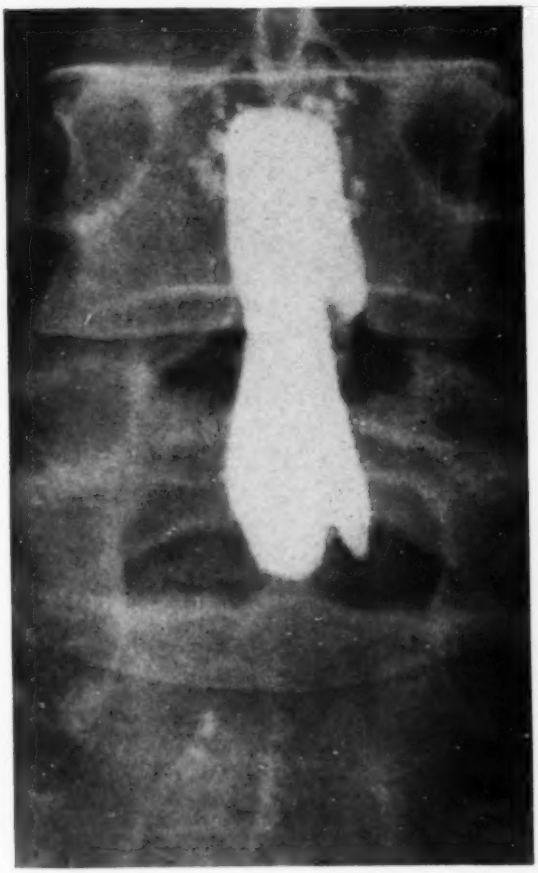

Fig. 35. Case R. S. "Amputation" of right fourth and fifth lumbar nerve root sheaths by lateral protrusions.

HERNIATIONS OF CERVICAL INTERVERTEBRAL DISCS

Scant consideration has been given in past years to herniations of cervical intervertebral discs. Recent studies, however, indicate their relatively common incidence and their importance in explaining cases of heretofore obscure brachial neuralgia and neuritis. In former years such herniations

Fig. 37. Case G. S. Recurrent fourth lumbar herniation with distortion of the canal and nerve root sheaths.

Fig. 38. Case R. F. Narrowing of the fifth cervical interspace with osteophytes about the vertebral bodies. Myelogram of this case is shown in Figure 42.

Fig. 39. Case M. F. Oblique projection demonstrates narrowing of the left sixth intervertebral foramen with osteophytes about its margins. Myelogram is shown in Figure 43.

Fig. 40. Cervical myelogram following injection of 6 cc. of pantopaque by the lumbar route, with filling of the lower five cervical nerve root sheaths. While a questionable slight deformity was present in the region of the left sixth cervical root sheath, clinical findings were not sufficiently conclusive to warrant exploration.

usually were not differentiated from spinal cord tumors, either clinically or pathologically. The lesions removed were considered to be extradural chondromas by Elsberg[2] and other observers.

In 1942, Semmes and Murphey,[7,8] described a syndrome characterized by pain in the neck radiating to the shoulder, precordium and arm, and by sensory changes in the index and middle fingers on the affected side. Bucy and Chenault[1] in 1944 reported a case of compression of the seventh cervical nerve root by herniation of an intervertebral disc.

In 1944 Spurling and Scoville[10] reported a group of patients with radicular pain in the upper extremity due to lateral rupture of the fifth or sixth cervical intervertebral

Fig. 42. Case R. F. Lipiodol cervical myelogram. In spite of globulation of oil, a large lateral and midline defect is visualized above the sixth cervical vertebra on the left. A moderately large protrusion was found compressing the sixth cervical nerve root.

disc. They introduced the "neck compression" test. By tilting the head and neck toward the side of the lesion, the patient's radicular pain is reproduced. Pressure on top of the head increases the pain. This test has proved to be of much value in differentiating cases of brachial neuralgia due to lateral herniations of the cervical intervertebral discs from those due to other causes.

In more than 50 per cent of all cases of herniations of cervical discs, lateral and oblique roentgenograms of the cervical spine demonstrate localized narrowing of the affected disc (Fig. 38) and osteophytes about the margins of the vertebral bodies

Fig. 41. Case A. B. Questionable minimal defect involving the right sixth nerve root sheath above the body of the sixth cervical vertebra. This patient was not explored but clinical and roentgenological findings suggest a small lateral protrusion.

Fig. 43. Case M. F. Pantopaque myelogram demonstrating lateral defect involving the left sixth cervical nerve root sheath. A protrusion was found at operation.

and intervertebral foramina (Fig. 39). Pantopaque myelography provides an excellent method of confirming the diagnosis and of localizing accurately the site of the protrusion.

Fig. 44. Case B. O. Pantopaque myelogram demonstrating a defect above the seventh cervical vertebra on the right. At operation a moderately large herniation of the disc between the sixth and seventh cervical vertebrae was found with compression of the seventh cervical nerve root.

In the normal cervical myelogram, filling of the nerve root sheaths appears to be more satisfactory with pantopaque than with other contrast media. Small lateral herniations cause a failure of filling of the nerve root sheath on the side of the lesion. Large herniations produce defects in the opaque column as well as in the affected nerve root sheaths.

Four cases of herniation of cervical intervertebral discs were operated upon in this series (Fig. 14, 42, 43 and 44). As noted above, one patient was believed preoperatively to have an extramedullary tumor; the other 3 cases were diagnosed correctly. Figures 40 and 41 demonstrate minimal defects of doubtful significance; both of these patients had clinical findings suggestive of herniations but were not explored.

Complete blocks are rare and none were found in this series. Such patients rarely have radicular pain or paresthesia alone and usually present symptoms of spinal cord compression. Figure 45 is a reproduction of a roentgenogram (not in our series) which demonstrates a complete block. This patient had a quadriplegia and was thought to have a cervical spinal cord tumor. Two cubic centimeters of iodized oil introduced into the cisterna magna were arrested opposite the fifth cervical intervertebral disc. At operation, a large disc herniation was found.

Fig. 45. Complete cervical block demonstrated by lipiodol introduced by cisternal puncture. Disc found at operation. (Case not in this series.)

Differentiation from Spinal Cord Tumors

It is difficult and frequently impossible to differentiate a large disc herniation from a spinal cord tumor, especially the extramedullary type. While a tumor may be situated anywhere, almost all herniations

are noted opposite intervertebral discs, anterior or lateral to the subarachnoid space of the spinal canal; rare exceptions are noted when herniations become detached and lie free in the epidural space (Fig. 26). A tumor may erode vertebral bodies and pedicles; a herniation never does.

PANTOPAQUE AS A CONTRAST MEDIUM

In comparing pantopaque with other contrast media, it is our impression that there are certain definite advantages in the use of pantopaque over other available substances with few discovered disadvantages.

An ideal contrast medium should have the following characteristics: (1) it should be sufficiently radiopaque or radiolucent to offer moderately high contrast to surrounding tissues; (2) it should be non-irritating and non-toxic; (3) it should be absorbed completely without evidence of pharmacologic effect on any body tissues; or (4) if not absorbed, it should have physical characteristics which allow it to be removed completely and easily; (5) it should have the proper density and viscosity to allow it to flow readily in a homogeneous stream over the dependent portions of the subarachnoid space of the spinal canal and to fill the axillary pouches and sheaths of the spinal nerve roots; and (6) it should be of such a nature that it can be used to visualize all portions of the spinal canal.

To date, no contrast substance has been developed which meets all requirements of an ideal medium.

The use of air and oxygen for myelography obviates the objection to an irritating and non-absorbable medium. However, there are certain inherent disadvantages in their use. Considerable discomfort usually follows such injections; gas offers low contrast and minimal defects are not visualized in many cases. Gas is of no value in myelographic studies of the cervical and thoracic spine.

Thorotrast has had a very limited use in myelography. Thorium compounds are irritant and radioactive and most workers have been reluctant to inject a medium of this kind into the subarachnoid space.

Absorbable iodide compounds such as diodrast have been employed experimentally for myelography but have produced sufficient local irritation as to render them unacceptable for clinical application.

Lipiodol, first introduced for intraspinal use by Sicard and Forestier[9] in 1922, and for demonstrations of herniations of intervertebral discs by Mixter and Barr[5] and Hampton and Robinson[3] in 1934, has been the medium used most frequently in the past. It is highly radiopaque and flows fairly readily although somewhat sluggishly. While in the average case it flows in a homogeneous stream, it has a tendency to break up into small globules, especially if the stream of oil loses continuity in the course of injection or manipulation. Once this occurs, it is usually impossible to restore homogeneity to the pool. This property makes it especially unsuitable for cervical myelography.

Lipidol is absorbed very slowly, if at all, and if incompletely removed may lodge indefinitely in the ventricles, cisterns or subarachnoid space of the brain or in the spinal canal or along the spinal nerve root sheaths. While all or nearly all of the oil can be removed in most cases, it is sufficiently viscous so that injection and removal are frequently difficult.

There is some uncertainty regarding the toxic or irritating qualities of lipiodol. Mild immediate reactions have been observed in occasional cases and a few observers claim that retained lipiodol exhibits slightly irritant characteristics. Considering the fact that lipiodol had been used as a myelographic medium for years before a satisfactory technique was developed for removal, the number of cases reported in which there was alleged irritation by the retained oil have been very few indeed. Certainly it cannot have very marked irritant qualities or more conclusive evidence to that effect would have been produced by this time.

In general, however, lipiodol has proved to be a very valuable agent for myelography in spite of the disadvantages described above.

Pantopaque, a mixture of ethyl esters of isomeric iodophenylundecylic acids, is an oil type of fluid, colorless to pale yellow in appearance, with a specific gravity of 1.263 at 20° C. It contains 30.5 per cent of iodine in organic combination as compared with 40 per cent of iodine in lipiodol, and at 37.5° C. is one-seventeenth as viscous as lipiodol. Being of low viscosity it is injected and withdrawn with ease. There is less likelihood of pantopaque entering the epidural space as no force is required to inject it.

It moves up and down the canal more rapidly than lipiodol. Frequently, it breaks up into droplets; when re-pooled, it tends to flow together again more completely than lipiodol.

It appears to flow more readily into nerve root sheaths than lipiodol, thus outlining small defects more effectively.

While pantopaque in the subarachnoid space of animals exhibits moderate toxicity, no irritant or toxic manifestations have been observed in man in cases where the medium has been left in the spinal canal.

Ramsey, French and Strain[6] report that, if left in the subarachnoid space, pantopaque is absorbed at the rate of approximately 1 cc. per year. Wyatt and Spurling[13] studied 6 patients in whom 3.5 cc. of pantopaque was injected into the lumbar subarachnoid space and left in place. Subsequent roentgenographic studies were made of the skull and entire spine and estimates made as to the amount of residual pantopaque present. Quantities varying from about 0.1 to about 1.5 cc. were found to remain at intervals ranging from nine to fifteen months after injection. None of the patients had symptoms referable to the retained pantopaque.

In 41 per cent of our cases all of the pantopaque was removed at the conclusion of the myelographic examinations; in an additional 45 per cent of cases 95 per cent or more was removed; in 12 per cent, 30 to 95 per cent was removed; in 2 cases (less than 2 per cent), none of the pantopaque was removed. No ill effects were noted in any patient from whom the pantopaque was not completely removed.

RÔLE OF MYELOGRAPHY IN THE DIAGNOSIS OF HERNIATIONS OF INTERVERTEBRAL DISCS

The diagnosis of herniation of one or more intervertebral discs should be based primarily upon the clinical features of the case. A complete history and neurological examination will suggest the correct diagnosis in most cases. However, it is frequently impossible from clinical findings alone to localize precisely the protrusion or to rule out multiple protrusions.

A careful and complete roentgen examination of the spine should precede myelography, not merely to rule out anomalies and abnormalities but to give the examiner a picture, or map as it were, of the area which he is to study subsequently with contrast material. In the lumbar spine, abnormalities such as narrowing of an interspace, presence of osteophytes about the vertebral bodies adjacent to a herniated disc, or alterations in width of an interspace on anteroposterior and lateral flexion and extension are noted in a minority of cases. In the cervical spine, localized narrowing of an interspace with osteophytes about the margins of the vertebral bodies and intervertebral foramina are seen more frequently with a disc protrusion.

Pantopaque myelography should precede exploration in nearly every case for the following reasons:

(1) It offers objective evidence as to the presence of a lesion. It should be noted, however, that negative findings do not exclude the presence of herniations lateral to the limits of the subarachnoid space, which are found in a small minority of cases.

(2) It localizes the lesion and provides valuable information regarding the presence or absence of multiple herniations.

(3) The procedure is a simple, safe and nearly painless one.

(4) If pantopaque is left in the subarachnoid or epidural space, there is evidence that the retained substance is non-irritant, non-toxic and is absorbed slowly.

(5) While the operation for removal of a herniated disc is not formidable, it is sufficiently serious so that all information possible should be available before it is undertaken.

Myelography is of least value in patients with narrow spinal canals or with underdeveloped nerve root sheaths, especially if the herniation is small and laterally placed. Apparently high terminations of the caudal sac at or above the fifth lumbar disc level should be viewed with suspicion in patients with clinical evidence of herniation of a low lumbar disc.

COMMENT

Conflicting opinions by eminent neurosurgeons, roentgenologists, and orthopedists regarding the value of myelography in the diagnosis of protrusions of the intervertebral discs have resulted in confusion and bewilderment among the general medical public in the treatment of such cases. The introduction of an easily removable, non-toxic contrast medium has obviated most of the objections to contrast myelography. The injection and removal of pantopaque are only slightly more difficult than performance of a lumbar puncture. No untoward sequelae or complications referable to myelography with pantopaque have been noted.

Myelography should be done in nearly every case of low back pain with sciatic radiation before the patient is subjected to a fusion operation on the spine or sacroiliac joints. Thus many unnecessary and fruitless operations will be avoided. The removal of a herniated intervertebral disc from the lumbar or cervical region can be accomplished through a small incision and with minimal disturbance to the bony structures. Myelography provides sufficiently accurate localization so that the operative procedure may be limited to the site or sites of the lesions.

Pantopaque myelography occupies much the same place in the study of spinal lesions that pneumo-encephalography occupies in the study of cerebral disorders. Neither method replaces careful clinical study; both, however, are valuable adjuncts to neurologic diagnosis.

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Vol. 53, No. 4

ARTERIOGRAPHY FOR THE DEMONSTRATION OF INTRACRANIAL ANEURYSMS

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ALTHOUGH it is one hundred and seventy-five years since arterial aneurysms of the brain were first recognized at autopsy, relatively few cases are even now being diagnosed during life. Of these the majority are still disclosed during operation for other intracranial lesions; e.g., of aneurysms found on the basilar and vertebral arteries in reported cases by Dandy, 11 of 21 were disclosed during operations for trigeminal neuralgia and Ménière's syndrome.

In 1927, Egas Moniz[7] introduced arterial encephalography and in 1933 he reported the first demonstration of an intracranial aneurysm of the right internal carotid artery by means of cerebral arteriography, thus giving new impetus to the search. Since then, many cases have been diagnosed by this method. Although the injection of the internal carotid has usually been done directly following dissection of the vessel, other techniques have also been recommended. Direct puncture of the common carotid artery to obtain intracranial arteriograms was reported by Loman and Myerson[15] in 1936. Although direct puncture is not a formidable procedure, it requires much skill. The introduction of contrast material into the basilar artery can only be accomplished by the open operative techniques and this latter procedure has been adequately described in the paper by King.[12] It is our belief that the injection of the common carotid will not satisfactorily demonstrate the cerebral arteries since blood and the contrast medium will be shunted through vessels which do not directly supply the cerebral hemispheres. For this reason, dissection of the internal carotid on the side of the suspected lesion with introduction of the contrast material under direct visualization is advocated.

Although thorotrast has been generally used as the radiopaque medium for angiography, a state of doubt concerning its safety still exists. Because of the possibility that the injection of radiopaque thorium dioxide solution may cause toxic symptoms later, the use of thorotrast has been discontinued by many investigators. Others are not impressed by this possibility as a serious objection.[25] However, Northfield and Russell[18] have described histologic studies in 4 cases in which thorotrast was retained in the lumen of the walls of the cerebral vessels following angiography. Actual occlusion of vessels by thorotrast was suspected in one case and this must be considered a very serious criticism of this opaque medium. A similar criticism is also applicable to the use of umbrathor. In 1938, Ekström and Lindren[9] published reports which demonstrated cerebral thromboses in 60 per cent of the brains which came to necropsy after intracarotid injection of thorotrast. No reports of such untoward sequelae following the use of diodrast have been reported. More recently diodrast, neoskiodan, uroselectan and similar preparations made for excretory urography have been utilized in cranial arteriography. These substances are non-toxic in the doses used, are quickly excreted from the body and do not damage the intima of the vessel. The density of the shadow of diodrast and similar iodine containing compounds, when utilized for arteriography, while satisfactory is not as marked as that produced by colloidal thorium dioxide. While none of the untoward effects and potential dangers ascribed by Gross[10] to the use of thorotrast have occurred in our group of cases, the 50 per cent solution of diodrast compound is now being used. Because of the reactions described by Gross

following the use of a solution of 70 per cent diodrast, this concentration has not been used. The use of the 35 per cent diodrast did not produce satisfactory visualization of the cerebral circulation. Thorotrast has been used in the injection of 13 of our cases, while 50 per cent diodrast compound has been used in 2 additional cases. Numerous procedures have been devised for the obtaining of complete cerebral angiograms. Thus Egas Moniz devised a seriograph termed the "escamateador" which permitted the rapid exposure and removal of cassettes. In this manner the serial visualization of the arteries and veins of the cerebral hemispheres could be made. Sanchez-Perez has more recently devised a portable apparatus which could be adapted to any roentgenographic table. Most of the investigators utilizing cerebral angiography have contented themselves with the making of single or double exposures. The techniques of the special devices suggested by Egas Moniz[8] and Sanchez-Perez[20] require special devices and these may not be available. In our cases, the surgeon has requested that the procedure be completed in the operating room and, for this reason, a simplified technique utilizing a portable roentgen apparatus was devised. Complete and satisfactory angiograms can be made by this method under sterile operating techniques. In addition, should further operative procedure be necessary, these may be completed after the viewing of the angiograms.

With the exception of the isolated reports such as those by Schüller,[21] Lodge, Walker and Stewart,[14] Zollinger and Cutler[26] and the very comprehensive reports by Sosman and Vogt[22] and Albright,[1] arterial aneurysms of the circle of Willis have received scant mention as problems in roentgen diagnosis. Pincherle,[19] in addition to crediting Schüller with the roentgenographic demonstration of an aneurysm of the internal carotid artery, stated that the reports by Schüller and by Speiss and Pfeiffer[23] were the only ones recorded in the literature up to the year 1922. In addition to the characteristic changes shown by the curved linear calcifications demonstrated in the supra- and parasellar area found in the walls of aneurysms, destructive changes should also be sought for in the margins of the sphenoidal fissures and in the carotid. However, there is nothing distinctive about the sellar destruction and sphenoidal fissure erosion. Such changes can be caused by tumors as well as aneurysms. Since the first visualization of an intracranial aneurysm by Egas Moniz, the graphic demonstration of other aneurysms by arteriography has been noted by other observers. In addition to demonstration of the internal carotid vessels, more recently the vertebral artery has also been injected by Takahashi[24] in 1940 and by King[12] in 1941. No angiographic demonstration of aneurysms in this vessel and its components has as yet been recorded. Injection of the vertebral artery produces better demonstration of the posterior communicating and posterior cerebral arteries. The use of arteriography to examine the basilar and vertebral arteries to exclude aneurysms is of special value for the following reasons: As yet the characteristic curvilinear calcifications found in the sellar region emphasized by Sosman and Vogt and by Dyke[6] have not been demonstrated in instances of the basilar or vertebral aneurysms because of the obscuring shadows of the petrous ridges and mastoid bones. Both Dyke and Dandy have suggested that encephalography could demonstrate aneurysms as definite intracranial lesions of uncertain identity.

Dandy[4] states in his excellent monograph that while important information is unquestionably obtained from angiography, it has also assumed an ever increasing though greatly overdone rôle in vascular lesions of the brain. However, when it is considered that in the group of aneurysms found in the basilar and vertebral arteries, 11 of the 21 cases were discovered during operative procedure for trigeminal neuralgia and Ménière's disease, the need for more accurate preoperative diagnosis becomes apparent. Similarly, aneurysms in

the carotid vessels have also been disclosed during operation for trigeminal neuralgia by Magnus[16] and Jefferson.[11] Zollinger and Cutler, Lodge *et al.*, and Albright noted instances where because of the symptomatology as well as the sellar deformity, the presence of an aneurysm has led to the preoperative roentgenological diagnosis of hypophyseal tumor, suprasellar tumor or cyst.

The entire procedure is carried out in the operating room so that there is no transporting of the patient and so that a completely sterile technique is adhered to throughout. A mobile apparatus is used and positioned so that lateral roentgenograms can be made since this view has proved to be of greatest value in the recognition of aneurysms. The factors used are as follows: The tube target distance is 35 inches, 80 kilovolts, 25 milliamperes, one-quarter second exposure. A Lysholm or Bucky-Potter diaphragm was not employed. A trial roentgenogram is made prior to the introduction of the contrast medium with the patient supine to determine if the position is correct and if the roentgen technique is satisfactory. This film is immediately processed so that whatever corrections are necessary are immediately undertaken. We have noted that film exposures made with a high speed technique employing a rotating anode tube have not proved as satisfactory as those made at slower speeds employing one-half or one-quarter second.

The technique employed for the visualization of the intracranial circulation by means of the injection of contrast medium into the internal carotid and the vertebral arteries is as follows: The dissection and visualization of the carotid sheath are carried out in the operating room by the supervising neurosurgeon. In the cases where injection of the vertebral artery is desired, the dissection is continued until the anterior scalenus muscle is seen. The sixth cervical vertebra is then found and the vertebral artery is freed from the surrounding tissues and followed to its origin from the subclavian artery. A broad catgut ligature is then passed under the artery in order to produce fixation and to temporarily occlude the vessel during the introduction and injection of the contrast medium. The adequacy of the collateral circulation by use of the Matas test, i.e., compression of the internal carotid artery in the neck, can be done upon the exposed vessel. A straight No. 18 gauge needle is introduced into the internal carotid, care being exercised to avoid puncture of the opposite wall. It is well to note the very excellent suggestion offered by King to inject the vertebral artery in which a No. 18 gauge lumbar puncture needle bent at right angles is used to introduce contrast material. In this manner the needle can be introduced along the same axis as the vessel well up into the vessel and yet the danger of puncturing the opposite wall is avoided. Following the preparation of the mobile roentgen apparatus, the needle is attached to a syringe containing 10 cc. of colloidal (thorium dioxide) thorotrast or 15 cc. of 50 per cent diodrast and is introduced into the vessel to be injected. At the beginning of the injection, the vessel is occluded below the inferior point of insertion of the needle by the previously placed ligatures. At a given signal, the contrast material is injected as rapidly as possible. When approximately three-quarters of the solution has been introduced, the first exposure is secured. A technician properly protected by lead gloves and apron and lead protective screen then makes rapid changes of the cassettes and three films are exposed at three second intervals. A final exposure is made at the end of twelve seconds. Careful coordination and a previous practice round have proved invaluable in this type of examination in order to secure adequate and complete angiograms. This procedure has been selected as the most favorable one after previous and other various techniques were employed. Previously, in several angiograms, a single lateral film only was made at the completion of the injection. Such a procedure is not adequate and it is possible that actual failure to demonstrate the aneurysm may

Fig. 1. Plain roentgenogram of the skull. This is made to determine if the position is correct and the roentgen technique satisfactory.

result. A case is described by Krayenbühl[13] in which an arteriogram was made and aneurysm was not disclosed but it was later demonstrated by operation.

A group of 15 arteriograms have been completed. One arteriogram was made in a case of suspected glioma and is excluded from this series. An attempt was also made to inject the internal carotid artery postmortem in a case where rupture of an aneurysm was suspected but permission for pathological examination of the brain could not be obtained. This group of cases with the roentgen aspects in a group of aneurysms as demonstrated by arteriography and the subsequent course following the injection of thorotrast will be discussed in a future paper.

In order to demonstrate the difficulties concerned in the diagnosis of an intracranial aneurysm, the case history of one of our series is given. It will be seen that although localizing disturbances were present and an aneurysm was suspected, it was necessary to consider other lesions.

REPORT OF CASE

M. C., female Italian, factory worker, aged thirty, was admitted to Grace Hospital on November 23, 1943. She was disoriented and complained of marked dizziness and left-sided headache. It was learned that one month prior to admission, she had complained of headaches that were extremely severe in the morning. Eleven days before admission while at work, she complained of a sudden severe pain in the head which was localized to the left frontal area and above the left ear. Brought to the restroom, she stated that she heard noises within her head. She appeared dazed and her eyes were deviated to the right. Her speech was unintelligible and she could not move her legs. Approximately fifteen minutes later, she had marked projectile vomiting. The next day, the symptoms persisted and she vomited several times. Nine days before admission, she improved slightly. She was able to stand and her speech was intelligible at intervals. Seven days before admission, she became markedly confused and complained of severe left-sided headaches.

In the previous medical history, the patient stated that she had had a chronic left otitis media for the past five years.

The physical examination demonstrated a completely disoriented female because of the motor and sensory aphasia and paraphasia. The pupils were round, regular, equal and reacted normally to light. Diplopia was present. The patient closed one eye for near vision. There was no evidence of nystagmus or exophthalmos.

Fig. 2. The contrast medium has been injected into the internal carotid and is seen outlining the carotid siphon. The aneurysm arises from the internal carotid artery near its junction with the middle cerebral artery. This is the type of aneurysm which is classified as the supraclinoidal type by Egas Moniz. Contrast medium in the original roentgenograms could be seen outlining the ophthalmic and anterior choroidal arteries. Three-quarters of the total amount of the contrast material has been injected.

The fundi showed some papilledema and the veins appeared distended.

A neurologic examination attempted on admission of the patient was not satisfactory and an additional complete examination was done a day later when the patient was more cooperative. In the examination of the eyes, there was no evidence of paralysis of the fourth and sixth nerves. The patient showed diplopia with marked ptosis of the left lid. There was anesthesia of the first and second branches of the right trigeminal nerve. The acuity of the eighth nerve on the left was diminished. There were

Fig. 3. The contrast medium is seen outlining portions of the arterial system. Small amounts are also noted in the venous tributaries. The exposure is made three seconds after the first exposure.

involuntary movements of the left arm and the right showed less motor power than that on the left. In addition, there was increased tonus of the right arm. Superficial abdominal reflexes were absent on the right and a Babinski could also be elicited on the right. The blood pressure was 110 systolic, 76 diastolic. Negative Wassermann and Kolmer tests were reported. Examination of the spinal fluid showed a slight xanthrochromia. The red cell count of the spinal fluid was 10,900, 190 monocytes were seen and there were 2 polymorphonuclear leukocytes. The total protein was 27 mg. per 100 cc. The lumbar spinal puncture revealed initial pressure of 340 mm. H_2O; 6 cc. of pink cerebrospinal fluid was removed for examination.

Because of a low grade fever, the presence of a brain abscess could not be excluded at this time and it was deemed advisable to rule out a brain tumor. Sulfadiazine was administered

Fig. 4. Large venous channels are outlined by the contrast medium. The aneurysm is still filled. This exposure is made six seconds after the first exposure.

to the patient. For sixteen days, the patient improved slowly. On the nineteenth hospital day, a complete Weber's syndrome was noted. The patient complained bitterly of a pounding left frontal headache. The temperature at this time was normal and sulfadiazine therapy was discontinued.

A neurosurgical consultant at this time suggested that an aneurysm or a tumor with hemorrhage could produce the syndrome. Ventriculography, however, revealed no evidence of displacement or deformity of the ventricles and no definite tumor mass could be demonstrated.

Fig. 5. Only a trace of contrast medium can be visualized in the venous system. It is still retained in the aneurysmal dilatation. Final exposure is made twenty-two seconds after the first exposure.

Some decalcification of the lesser sphenoidal wings was noted on the previous roentgen examination and the roentgenologist suggested that an attempt be made to exclude a vascular lesion in this area.

On the fifty-first hospital day, angiography was done, the neurosurgeon supervising the operative procedure. The collateral circulation was demonstrated to be adequate by the Matas test. The internal carotid artery was then injected under direct visualization using 10 cc. thorotrast (Fig. 1, 2, 3 and 4). A roentgenogram was made at the end of approximately twenty-two seconds in which contrast material could be seen remaining in the aneurysm (Fig. 5). The internal carotid was then ligated but not divided. There was marked improvement of the physical signs following the ligation and no complications ensued. The patient was discharged and has been followed for approximately two and one-half years. At the present time, the patient is entirely well.

Since the time of operation, she has been free of all of the symptoms. The papilledema and congestion of the retinal veins are no longer demonstrable. The patient is able to elevate the left upper lid so that the entire pupil can be exposed.

It is to be stressed that in this case the patient had refused to undergo any cranial operative procedure so that it was impossible to attempt clipping of the internal carotid intracranially as recommended by Dandy. The sudden severe pain complained of by the patient was apparently due to vascular expansion and possible rupture of the aneurysm. Roentgen examinations demonstrated no evidence of erosion of the clinoid processes although some decalcification of the lesser sphenoidal wings had been indicated. Ventriculography in this case demonstrated no defects in the region of the cisterna interpeduncularis and chiasmatis. Although the signs and symptoms became more marked as the patient remained in the hospital, apparently a diagnosis could not be satisfactorily arrived at when the patient was first admitted.

Adequate angiography will demonstrate the contrast material entering into the skull. In 70 per cent of cases, the carotid artery describes a complete "S" curve and then divides into the anterior and middle cerebral vessels. The term "carotid siphon" has been coined to designate the bends of the carotid artery before its branching. The middle cerebral artery in the majority of cases then divides almost immediately into the posterior temporal, the posterior parietal and the angulate gyrus. The anterior cerebral vessel passes forward and upward and has one important branch, the pericallosal which follows the outline of the corpus callosum. In several cases, the origin of the ophthalmic and the anterior choroidal vessels from the internal carotid with the ramifications in the orbit has also been noted. The later roentgenograms demonstrate the contrast material leaving the skull via the veins. It is interesting to note in our illustrations the manner in which the aneurysm is still outlined by the contrast medium although the major portion of the medium has left the intracranial vessels. In our cases, reference to the illustrations in the text by Egas Moniz has proved invaluable in the interpretation of angiograms.

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THE DISEASES OF THE VERTEBRAL COLUMN

A ROENTGENOLOGIC ANALYSIS*

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1. OBJECT OF THE STUDY

ANATOMICAL lesions of the spine have a high incidence but produce clinical manifestations in only a minority of cases. For instance, in a series of 1,824 vertebral lesions of all kinds, in which the correlations between clinical and roentgen findings have been analyzed during the past nine years, there were clinical signs and symptoms referable to the vertebral lesion in only 28 per cent of the patients. Common clinical manifestations of disease of the spine—viz., pain in the back and neck, limitation of vertebral movements, and evidence of nerve root involvement—may be caused by many different diseases other than vertebral, even in the presence of a vertebral abnormality, and the roentgenologic demonstration of a vertebral lesion does not prove the vertebral origin of the complaint. The question, then, is: Which are the lesions of the spine that do produce clinical manifestations, and why?

To answer this question, it is necessary to analyze the clinical findings, to analyze the roentgen findings, and to examine whether correlations can be established between them. The first step consists in defining the vertebral lesions as accurately as possible. The historical definitions, based upon entirely different principles, cannot be used for this purpose. Some order has to be found into which the phenomena can be grouped in accordance with the canons of classification.

If, for the purpose of this study, one regards the diseases of the spine as nothing but vertebral localizations of the diseases of bone and joints, the vertebral lesions would seem to fall naturally into four main groups:

1. Diseases of vertebral bone (spondyl-osteitis*)
2. Diseases of vertebral symphyses
3. Diseases of vertebral synovial joints (spondyl-arthritis*)
4. Diseases of vertebral ligaments

Since the characters of bone and joint disease vary to some extent with the localization, it is to be expected that these diseases will show some particular features when they involve the spine. It is the object of this paper to define the roentgenologic appearances of the four groups of vertebral diseases.

2. DISEASES OF VERTEBRAL BONE

The lesions of vertebral bone may be grouped according to certain dominant morphologic characters:

1. Diseases of vertebral bone
   a. Necrosing
   b. Predominantly rarefying
   c. Predominantly condensing and/or hypertrophic
   d. Lesions of growing bone
   e. Tumors

a. Necrosing Bone Lesions. Necrosis of vertebral bone is produced by the same causes as in other bones. Tuberculous and pyogenic infections are common causes; syphilis, brucellosis, mycoses, trauma, and roentgen irradiation may also induce necrosis. As in other bones, abscess formation may or may not develop, according to the variable factors involved.

As a rule, the vertebral bodies are involved much more frequently than the

* In current usage, "osteitis" means bone disease, and "arthritis," joint disease. The terms do not necessarily imply inflammation. Although obsolescent and equivocal, these designations are added here for the sake of conformity with the nomenclature generally adopted in the pathology of bone and joints—a conformity necessitated by the general trend of this study.

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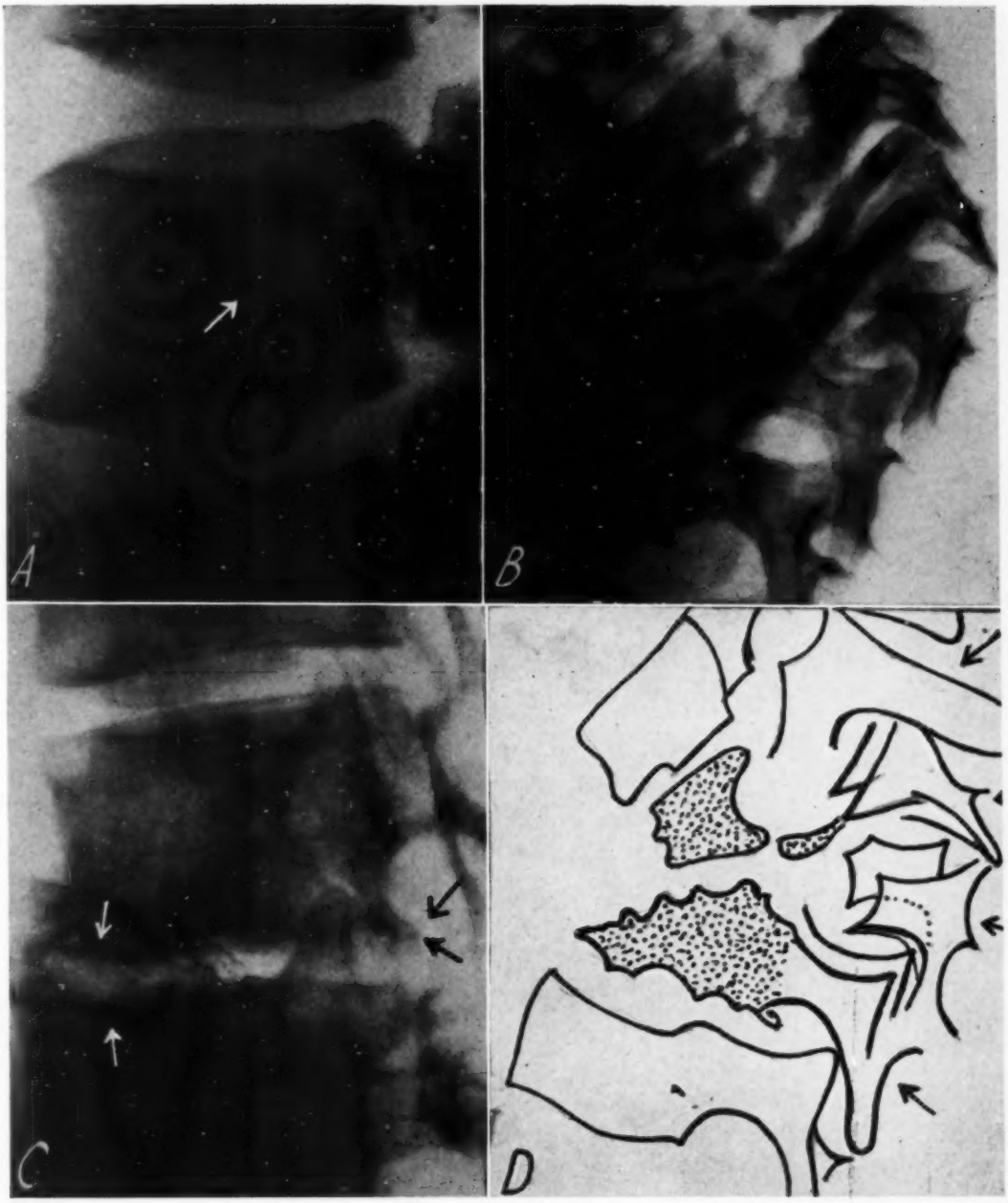

Fig. 1. Necrosing bone lesions. *A*, tuberculous necrosis confined to center of one vertebral body; duration, six years. No involvement of disc. *B*, roentgenogram, and *D*, tracing of case of tuberculous necrosis of vertebral bodies; the corresponding neural arches are nearly intact. *C*, tuberculous necrosis confined to marginal parts of contiguous vertebral bodies, with erosion of horizontal surfaces (white arrows), narrowing of disc space, and involvement of neural arch (black arrows).

neural arches. Apparently, the more abundant blood supply of the bodies favors invasion by blood-borne microbes, and the large venous sinuses in the bodies favor bacterial growth;[24] moreover, the cancellous vertebral bodies, once involved, are more easily destroyed than the compact neural arches. For instance, when tuberculous

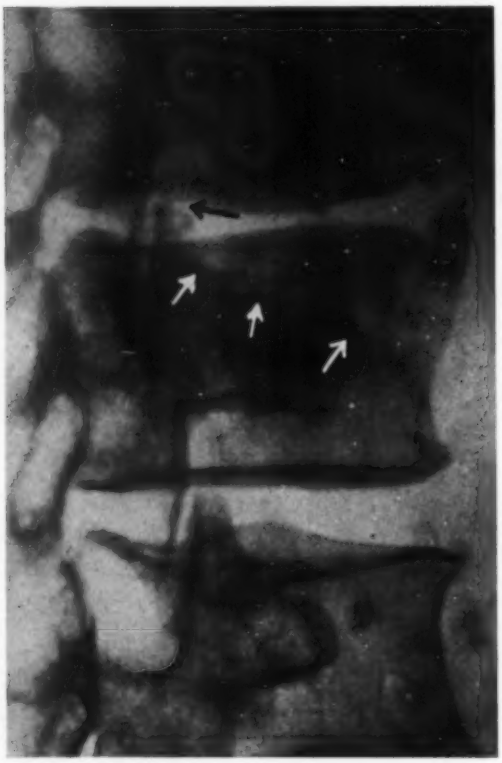

Fig. 2. Areas of recent destruction (white arrows) in a vertebral body showing condensation and osteophytes, the result of previous repair. Involvement of articular processes (black arrow).

necrosis takes a rapid course, the vertebral bodies become destroyed within a short time, but the neural arches remain intact and seem to point, as it were, to the empty spaces originally occupied by the bodies and discs (Fig. 1*B*). Necrosis may remain confined to the central part of a vertebral body (Fig. 1*A*), or to the marginal parts of contiguous vertebral bodies (Fig. 1*C*). The variable involvement of the vertebral discs in these cases will be discussed under heading 3 presently.

Low grade tuberculous and pyogenic infections may spread from the vertebral body into the neural arch[10,11] and erode it (Fig. 1*C* and 2); but necrosis beginning in, and confined to, the neural arch is rare. When present, it is usually caused by pyogenic infection,[25] but only exceptionally by tuberculosis.[23]

The necrosed bone may become replaced by new bone arising from adjacent areas. This repair is delayed and may even fail to occur when the infection is protracted, perhaps because the osseous connective tissue becomes fibrous. When infection is recurrent, new areas of necrosis may appear in regions showing signs of previous repair (Fig. 2). As a rule, bone repair is less definite in the neural arch than in the vertebral body. For instance, callus formation is usually absent or scanty in fractured neural arches and their processes. Only in the presence of low grade chronic disease does the neural arch form new bone, e.g., in Paget's disease. In this respect, the neural arch resembles the bones of the calvarium, to which it is related embryologically.

b. Predominantly Rarefying Bone Lesions. The vertebral bone may take part in systemic rarefaction, or demineralization, of the whole skeleton; or it may be involved by localized, or regional, rarefaction. Rarefaction is a reaction of bone to a large variety of etiologic stimuli and is not characteristic of any particular disease or group of diseases.

Since the cancellous vertebral body contains percentually less minerals than the compact neural arch, the bodies become rarefied more readily and rapidly than the arches. Indeed, systemic rarefaction of the whole skeleton often becomes evident in the vertebral bodies first.

Owing to the particular structure of the spine, certain secondary changes occur and are common to all types of vertebral rarefaction. Rarefied bone yields to pressure. The central perforated part of the horizontal surface of the vertebral body (lamina cribrosa) is structurally the weakest part of the vertebra and sustains the greatest statical stress.[21] Consequently this part rarefies especially easily and usually yields first. The adjacent disc, or a part of it, may then protrude into the vertebral body at this site;[20,21] this is known as intraspongious, or intravertebral, herniation of the disc, and as Schmorl's nodule (Fig. 3*C*). When rarefaction progresses, or is more severe, the entire horizontal surface may

yield to the pressure exerted by the expansible disc; this results in a semiglobular depression of the horizontal vertebral surface giving the appearance known as "fish vertebrae" (Fig. 3). Since, as a rule, the arches do not become rarefied as rapidly and severely as the vertebral bodies, they remain nearly intact while the bodies undergo compression. This leads to kyphosis in the thoracic spine, often associated with compensatory lordosis of the cervical and lumbar regions.[7,21,23]

In general, the degree of rarefaction corresponds roughly to the severity of the morbid stimulus; but sometimes rarefaction is out of proportion to the stimulus. In these cases an unnoticed trauma, or an untraceable infection,[7] may cause a vertebral body to become severely rarefied and to collapse. Histopathologic findings[8] suggest that this is usually associated with pronounced hyperemia of the involved bone. Possibly the lesions known as Kümmell's disease, Calve's vertebra plana, and certain types of adolescent kyphosis are merely different forms of the same basic disorder.

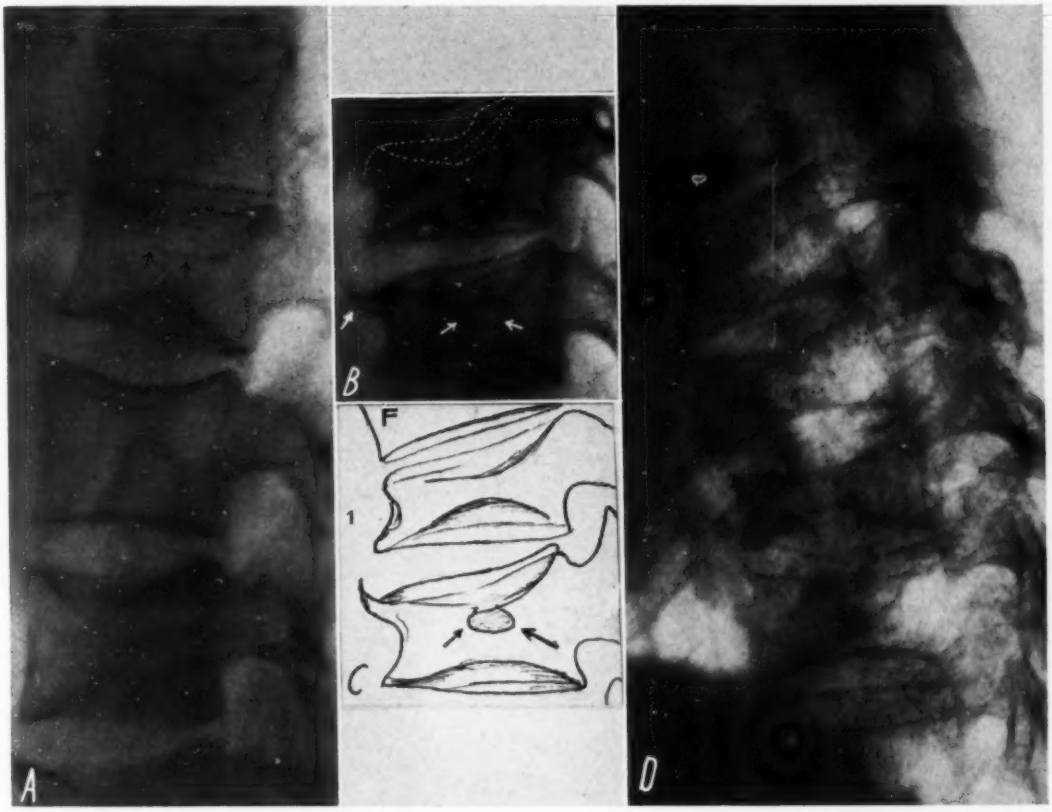

Fig. 3. Rarefaction. *A*, the lower vertebrae are held apart by the expanded discs, but the upper rarefied vertebrae have engulfed the expanded discs (black arrows), thereby producing narrowing of the intervertebral spaces and contact between vertebrae in spite of normal discs. *B*, roentgenogram, and *C*, tracing of a case with intravertebral herniation of disc (arrows) combined with expansion of the whole disc into the rarefied bone. *D*, compression of vertebrae and expansion of discs in a case of rarefaction of endocrine origin.

c. Predominantly Condensing and/or Hypertrophic Bone Lesions. Condensation and overgrowth of bone are both the result of production of new bone. New bone is coarser and denser than the original bone, as exemplified by bony callus. New bone formed within a bone is usually designated as condensation; and new bone formed

about the margins of a bone, as bone hypertrophy, exostosis, or osteophyte. Fundamentally, condensation and/or hypertropho are secondary reactions, preceded in most cases by rarefaction or necrosis; but they may be dominant features, and the preceding rarefying phase may escape observation. According to the amount of condensation, the involved areas appear either coarse (as in Paget's disease) or very dense (as in marble bone disease). Condensation may be systemic (fluorine poisoning, certain tumor metastases), or localized and caused by infectious, neoplastic, and traumatic lesions.

Systemic condensation involves the vertebral bodies and neural arches equally and simultaneously in most instances,[11] and seems to be the one disease in which the arches are as severely affected as the bodies. Tumor metastases often cause condensation in the vertebrae, but rarefaction and destruction in peripheral bones. Since condensation is usually a consequence of rarefaction—*un témoin de la raréfaction*, as Leriche and Policard put it—it is conceiv-

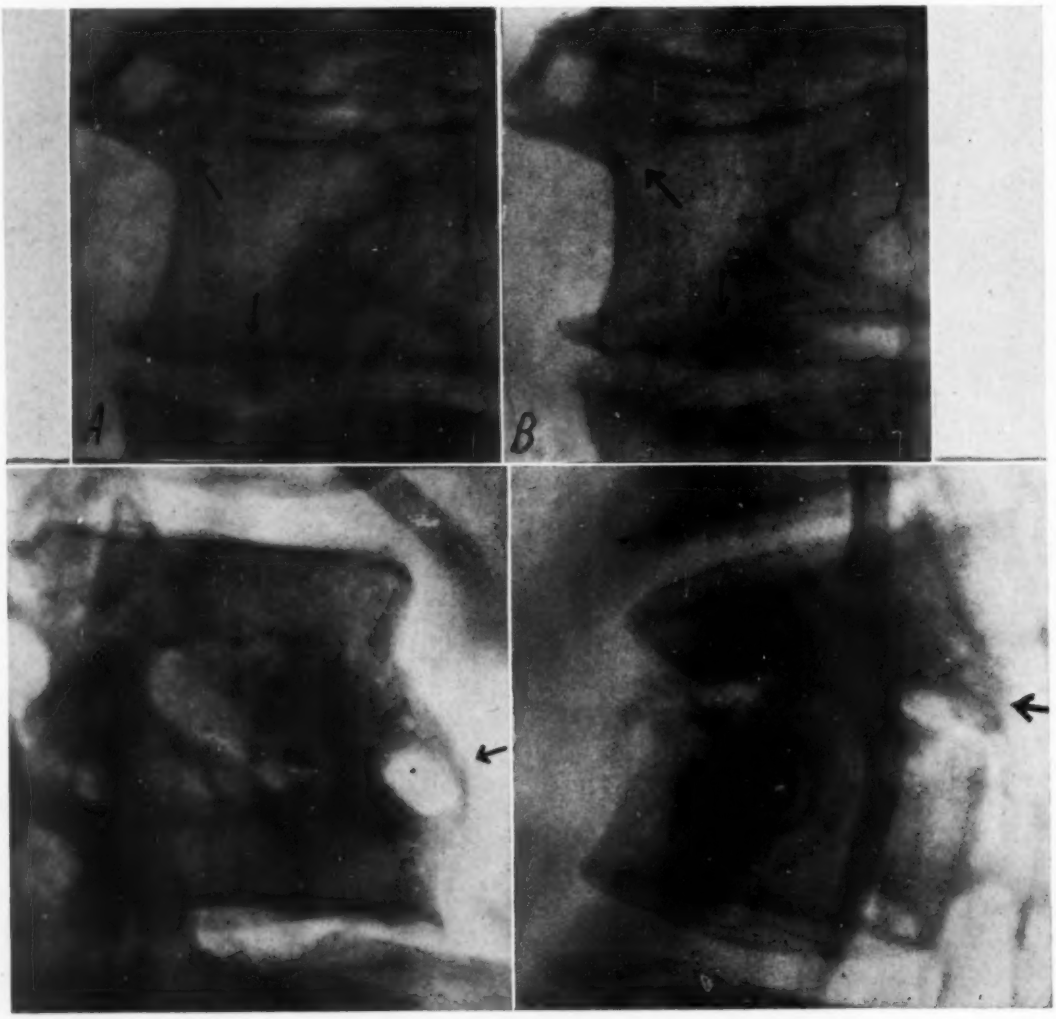

Fig. 4. Involvement of vertebral symphysis. Progressive erosion of vertebral margin by infection of undetermined origin, probably undulant fever. There is a three months' interval between *A* and *B*. Note progress of blurring and of partial dissolution of contours in *B* (arrows). Disc space narrowed, ligaments calcified. Lower row: obliteration of symphysis in the presence of bone necrosis.

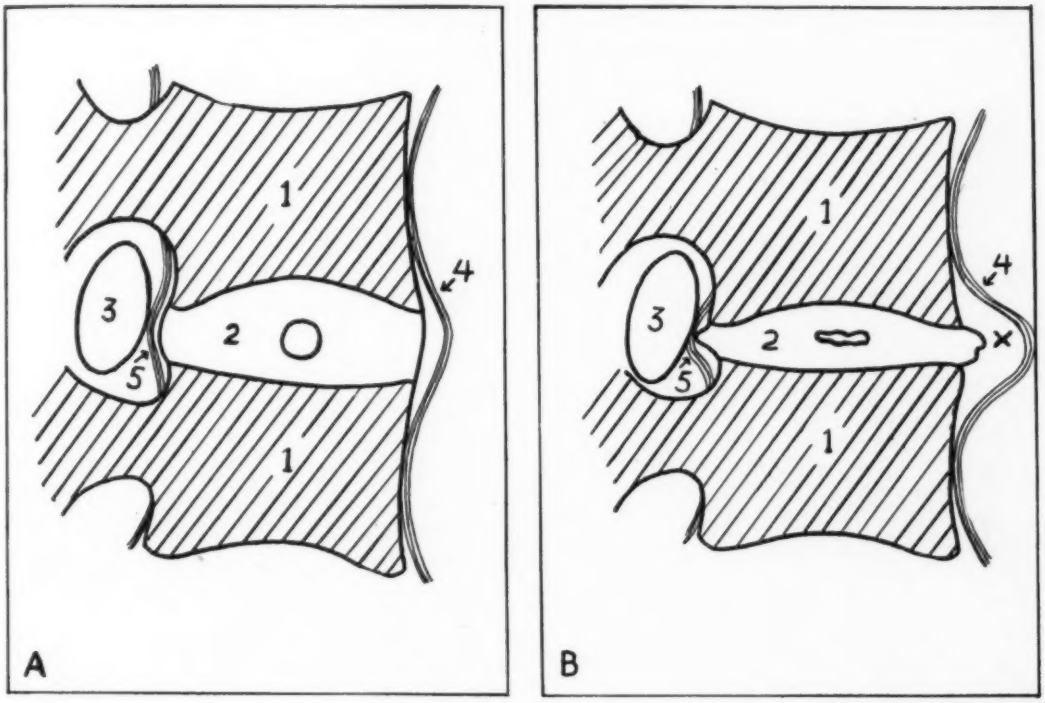

Fig. 5. Semidiagrammatic drawings showing the free space at the vertebral edge (*x*) into which new bone can grow (see text). *A*, normal, and *B*, in the presence of a thinned disc. *1*, vertebral bodies. *2*, intervertebral discs. *3*, cross section of nerve root. *4*, anterior longitudinal ligament. *5*, posterior longitudinal ligament. Note increase of free space and decrease of width of neural foramen caused by disc thinning, in *B*.

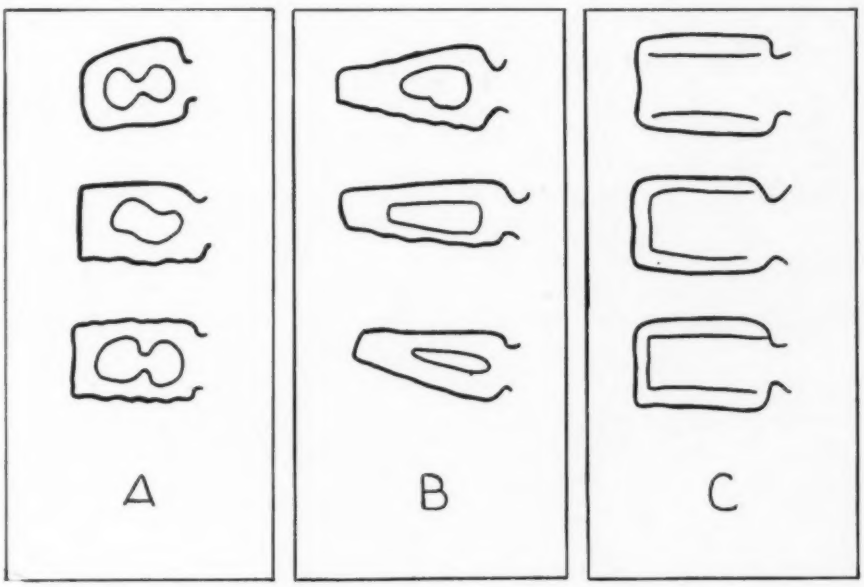

Fig. 6. Tracings of roentgenograms of lumbar spine of a boy, aged six, with severe rickets. The contrast between bone and soft tissues in the original roentgenograms is so poor that reproductions cannot be made satisfactorily. *A*, beginning compression of vertebrae, with clover-leaf shaped defects caused by enlargement of marrow spaces. *B*, three months later, the vertebrae have become thin bone plates separated by very wide interspaces. *C*, healing stage, with ossific lines causing double contours of vertebral bodies.

able that condensation is so common in the vertebrae because rarefaction also is often more pronounced in the spine than in other parts of the skeleton. Possibly the great vascularity of vertebral bone is responsible for this reaction.

Fig. 7. Osteochondrodystrophy.

Bony overgrowth of vertebrae usually occurs along the edge where the horizontal and vertical surfaces meet. These marginal osteophytes, often designated as "lipping," "spiculation," and "osteo-arthritis" of the spine,[7] are generally considered a distinctive morbid entity.[7,10,20,21,22] The findings in this series do not bear out this view. New bone is incapable of growing through normal differentiated connective tissue. Thus benign osteoma develops only where gaps are present in the periosteum.[2] Although devoid of a periosteum,[21] the vertebral body is surrounded by connective tissue (ligaments and cartilages) except along the vertebral edge. At this site the longitudinal ligaments separate from the vertical vertebral surface before bridging the disc. Consequently there is a triangular space free of connective tissue between bone and ligament, encircling the vertebral edge (Fig. 5). This, then, is the only space into which new bone can grow,[13] unless the ligaments and/or cartilages are torn or destroyed. Consequently osteophytes are usually confined to the vertebral edge no matter what their cause; they develop at this site in the presence of such different lesions as fracture, pyogenic infection, and as a result of the mechanical stimulation produced by thinning of the intervertebral disc (Fig. 2, 4, 5, 10 and 15).

d. Diseases of Growing Bone. The lesions arbitrarily included under this heading are a heterogeneous group, having in common only their occurrence in juvenile bone. This group is represented here by rickets, osteochondrodystrophy, and so-called adolescent osteochondritis; but many other lesions also belong in this category.

Rickets of the spine differs from rickets of peripheral bones because of the absence of primary epiphyses in the vertebrae. Consequently, rickets of the spine shows the rachitic lesion in a simplified model, so to speak.[16] Rarefaction causes the vertebral bodies to become compressed, and the enlargement of the medullary space, which is characteristic of rickets,[7] produces central defects in the vertebral bodies.[16] Owing to the associated muscular hypotony the physiologic curves of the spine become lost, to the extent that the lumbar lordosis may be reversed into kyphosis.[23] As in peripheral bones, lines of increased ossific density appear during the healing stages and produce a double contour of the vertebral bodies (Fig. 6).

Osteochondrodystrophy causes the same appearances in the spine as in other bones

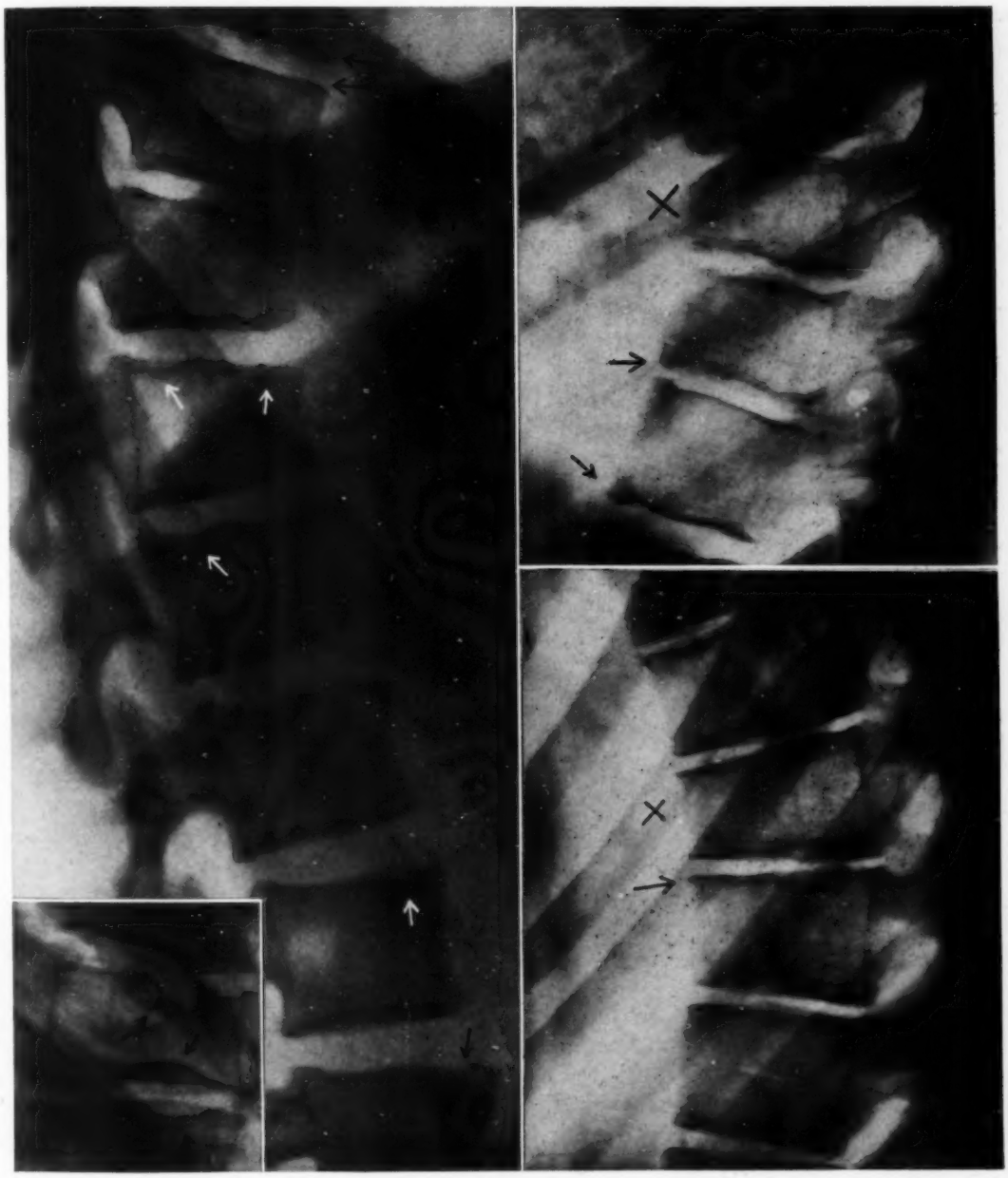

Fig. 8. Adolescent "osteochondritis," or "epiphysitis." The thoracic vertebral bodies are wedged as a result of rarefaction and compression, with scalloping of their horizontal surfaces as a result of protrusion of discs (white arrows). Normal annular epiphyses are present at the uppermost vertebral edges (horizontal black arrows). An abnormal epiphysis, showing fragmentation and surrounding condensation, is seen in the lowest vertebra (vertical black arrow). Insert shows compression of seventh thoracic vertebral body due to protrusion of disc in another case of adolescent osteochondritis. Roentgenograms on the right show osteochondritis at active stage (upper figure) and healed stage (lower figure).

(Fig. 7). There are no characteristic roentgenologic differences corresponding to the clinical varieties of the disease.

Adolescent osteochondritis—of which Perthes' disease is the best known example—occurs in the spine, as in other bones, at the time when the secondary ossific centers ossify and merge with the main parts of the

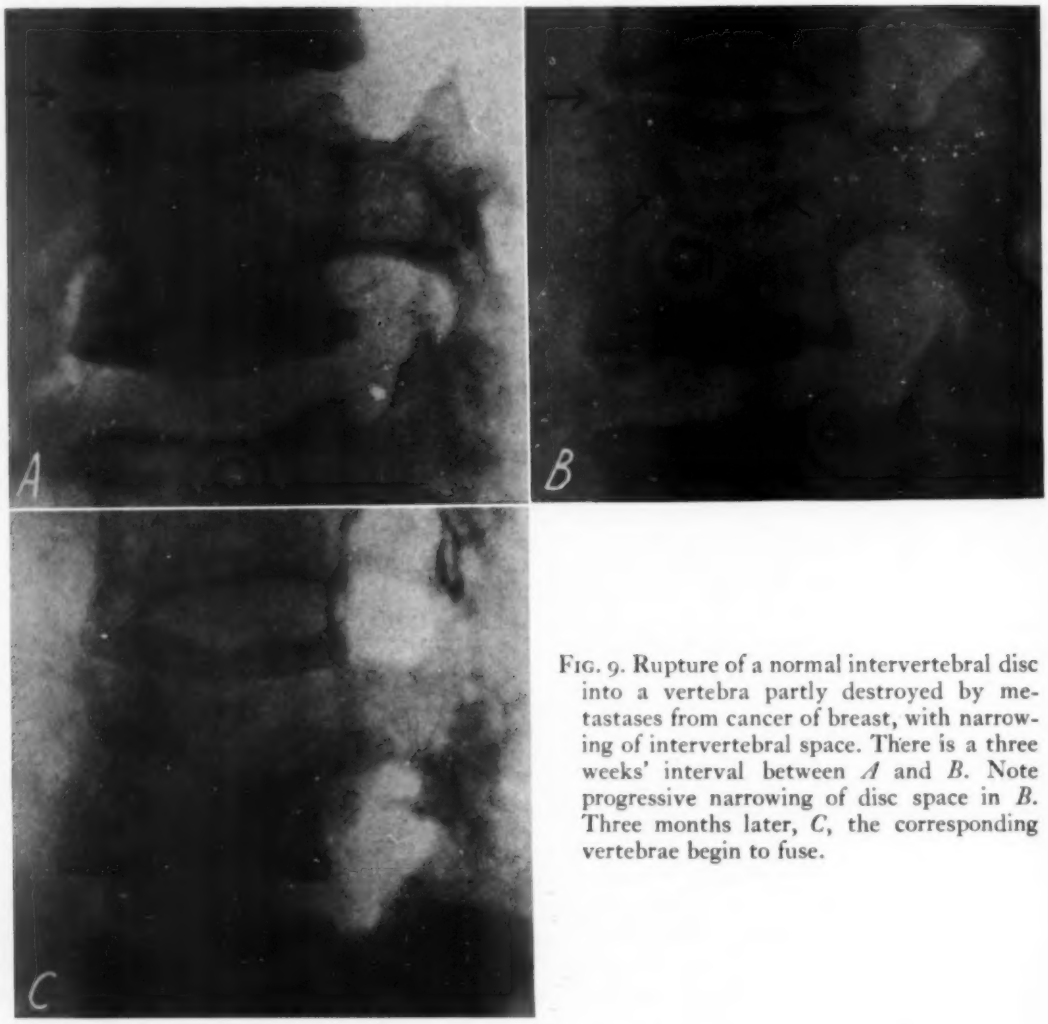

Fig. 9. Rupture of a normal intervertebral disc into a vertebra partly destroyed by metastases from cancer of breast, with narrowing of intervertebral space. There is a three weeks' interval between *A* and *B*. Note progressive narrowing of disc space in *B*. Three months later, *C*, the corresponding vertebrae begin to fuse.

bones. In the spine, these centers, or apophyses,[21] are ring-shaped and encircle the vertebral edge; when ossified and united with the vertebrae, they form the permanent vertebral edges of the adult.[21] As in the femoral head, osteochondritis produces fragmentation and deformity of these epiphyses. In the lumbar spine the involved epiphyses become easily detached from the anterior margins of the vertebral bodies where the tension of the longitudinal ligaments is strongest; condensation may develop as a secondary reaction (Fig. 8). Almost invariably there is an associated moderate rarefaction of all the vertebral bodies, with wedging of the bodies at the vertex of the thoracic kyphosis, and with protrusion of discs into the yielding bones. As a rule, the lesion of the epiphyses ("vertebral epiphysitis") is more definite in the lumbar spine than in other regions, while wedging of vertebrae ("adolescent kyphosis," Scheuermann's disease) is confined to the thoracic segments; but the two coexist in practically all cases and seem to represent merely different manifestations of the same disease (Fig. 8).

e. Bone Tumors, Primary and Metastatic. Primary bone tumors are rarer in the spine than in most other parts of the skeleton. Angioma, multiple myeloma, giant cell tumor, and Ewing's tumor were recorded in this series. The appearances are well known[19] and need not be discussed here. Tumor metastases have a high incidence in the spine, apparently because of the pecu-

liar topography of the vertebral veins. As already mentioned, osteoplastic (condensing) metastases are especially common in the spine. Contrary to current opinion, the disc spaces may become narrowed in the presence of vertebral metastases as a result of protrusion of the disc into the destroyed bone (Fig. 9). Involvement of the spine by Hodgkin's disease, the leukemias, and the lipoidoses is regarded here as metastatic.

Comments. Traumatic bone lesions have not been included, since their appearances are well known. The cystic and fibrosing bone lesions have been omitted because their classification is still debated. For the purpose of this study, the bone lesions had to be classified according to purely roentgenologic criteria, irrespective of clinical considerations. This classification, therefore, is not meant to supersede other accepted classifications based on different principles.

3. DISEASES OF VERTEBRAL SYMPHYSES

The articulation formed by two vertebral bodies and the intervening disc is an amphiarthrosis or symphysis according to anatomical texts, and may be designated as vertebral symphysis in order to distinguish it from the synovial joints of the spine.[14] Since every junction of two bones, or of bone and cartilage (including the cranial sutures and the cartilaginous epiphyseal junctions), is considered a joint anatomically, one would expect that lesions of symphyses be grouped with joint disease, or arthritis; but by definition, arthritis is a lesion of only the synovial joints. Since the vertebral symphyses have no synovial membranes, no joint cavities, no free articular cartilages, nor any other parts characteristic of synovial joints, the symphyses cannot be the seat of arthritis. This point is not merely academic, but has clinical implications which will be discussed under heading 6.

There seem to be two main groups of lesions of the vertebral symphyses:

2. Diseases of vertebral symphyses
   a. Originating in the vertebral bone
   b. Originating in the vertebral disc

a. Lesions Originating in Vertebral Bone. It is held that disc cartilage is impervious to infection and invasion by tumor cells;[20,21] yet there is no doubt that the disc often deteriorates as a result of infection of an adjacent vertebra.[7] Although resistant against tuberculous infection,[1,20] the disc usually becomes necrosed in the presence of tuberculosis of the corresponding vertebral body.[7,23] The records in this series suggest that the disc remains intact, at least for many years (Fig. 1*A*), when the infection is confined to the central part of the vertebral body; but whenever infection, no matter of what kind (Figs. 2 and 4), involves the vertebral horizontal surfaces, or the regions close to them,[15] the disc grows flat (Fig. 1*C*). Since during adult life the disc receives its nutrition exclusively from the vertebral body, through the lamina cribrosa,[21] the disc may become involved in these cases because its nutrition is, or was, impaired as a result of the lesion of the underlying bone.[15]

When congenital gaps,[21] traumatic fissures,[20] rarefaction, or necrosis of the horizontal vertebral surface allows parts of the disc to herniate into the vertebral body, proliferation of connective tissue often develops in both the bone and the disc and may gradually invade large areas or all of the disc.[21] This proliferation, being irreversible in cartilage, leads to fibrosis of the involved parts of the disc. When the fibrosed areas are large, loss of volume and of elasticity of the disc is the result, and the disc flattens down.

b. Lesions Originating in the Vertebral Disc. Owing to acute trauma, or to the recurrent slight injuries produced by the ordinary wear and tear of posture and motion, a number of "degenerative" changes may occur in the disc, such as fissures, rupture of disc cartilage or of the nucleus pulposus through the external disc layers, and detachment of fibrocartilage.[21] All these lesions lead, at least potentially, to increasing fibrosis of the disc eventually,[20] and the disc becomes flat. Since cartilage cannot be effectively regenerated, the lesion is permanent.

Fig. 10. "Degenerative" flattening of lumbar disc in a patient with severe sciatic pain. Note marginal osteophytes conforming to shape of free space at vertebral edge, and narrowing of neural foramen. Compare with Figure 5.

Flattening of the disc, no matter of what origin, induces a number of secondary alterations.[15]

c. Secondary Changes. At the level of a flattened disc the distance between the vertebrae diminishes proportionally. Consequently the corresponding neural foramina grow narrow (Fig. 5, 10 and 15), and the nerve roots which pass through them may become compressed,[5] especially in the cervical and lumbar segments, where the nerve roots are thick.[15] At the same time, the articular processes approach each other and may become subluxated,[4] with the result that their articular cartilages sustain increased stress. Erosion of these cartilages may develop and be followed by reactive changes in the corresponding apophyseal joints.[10] Swelling, thickening, and calcification of the apophyseal joint capsule may then occur and add to the compression of nerve roots in the already narrowed neural foramina.[11] Independently, but sometimes simultaneously, the edges of the vertebral bodies may come in contact during certain movements, since the discs, which are their natural buffers, are thinned; whereupon minute but recurrent fractures may occur in the vertebral edges,[21] and each fracture may heal with bony callus formation. As the injury recurs, the callus formations multiply, grow larger, and amount finally to osteophytes along the vertebral edges.[13] The common ligaments at the level of a flattened disc grow loose of necessity, owing to reduction of the intervertebral space which they bridge (Fig. 5). Consequently they bulge around the vertebral edge and increase the width of the triangular free space at this site (Fig. 10), thereby allowing osteophytes to grow particularly large (Fig. 15). These various changes, generally but erroneously termed "osteo-arthritis of the spine," may be designated as "discogenetic"—that is, produced by involvement of the disc[15]—in order to differentiate them from similar changes which are due to some other cause.

d. Narrowing of the Intervertebral Space without Involvement of the Disc. As a rule, narrowing of the space between contiguous vertebral bodies is caused by flattening of the intervening disc; but it may occasionally be the result of bone disease not involving the disc. In the presence of tuberculous and of pyogenic infections of the vertebrae,[1] of severe rarefaction (Fig. 11), and of certain tumor metastases (Fig. 9), the disc may remain intact but protrude, by its own expansibility, into the softened or destroyed bone. When the disc is completely expanded but the bone continues to yield to the weight of superincumbent parts, the softened bone may finally engulf the disc, and contiguous vertebrae may then come in contact (Fig. 3*A*) and become fused along their edges.[7]

Comments. The histopathologic varieties of disc "degeneration" have not been classified since they cannot be distinguished roentgenologically. Rupture of the disc into the spinal canal, although clinically significant, is included along with the lesions originating in the disc. Displacement or frag-

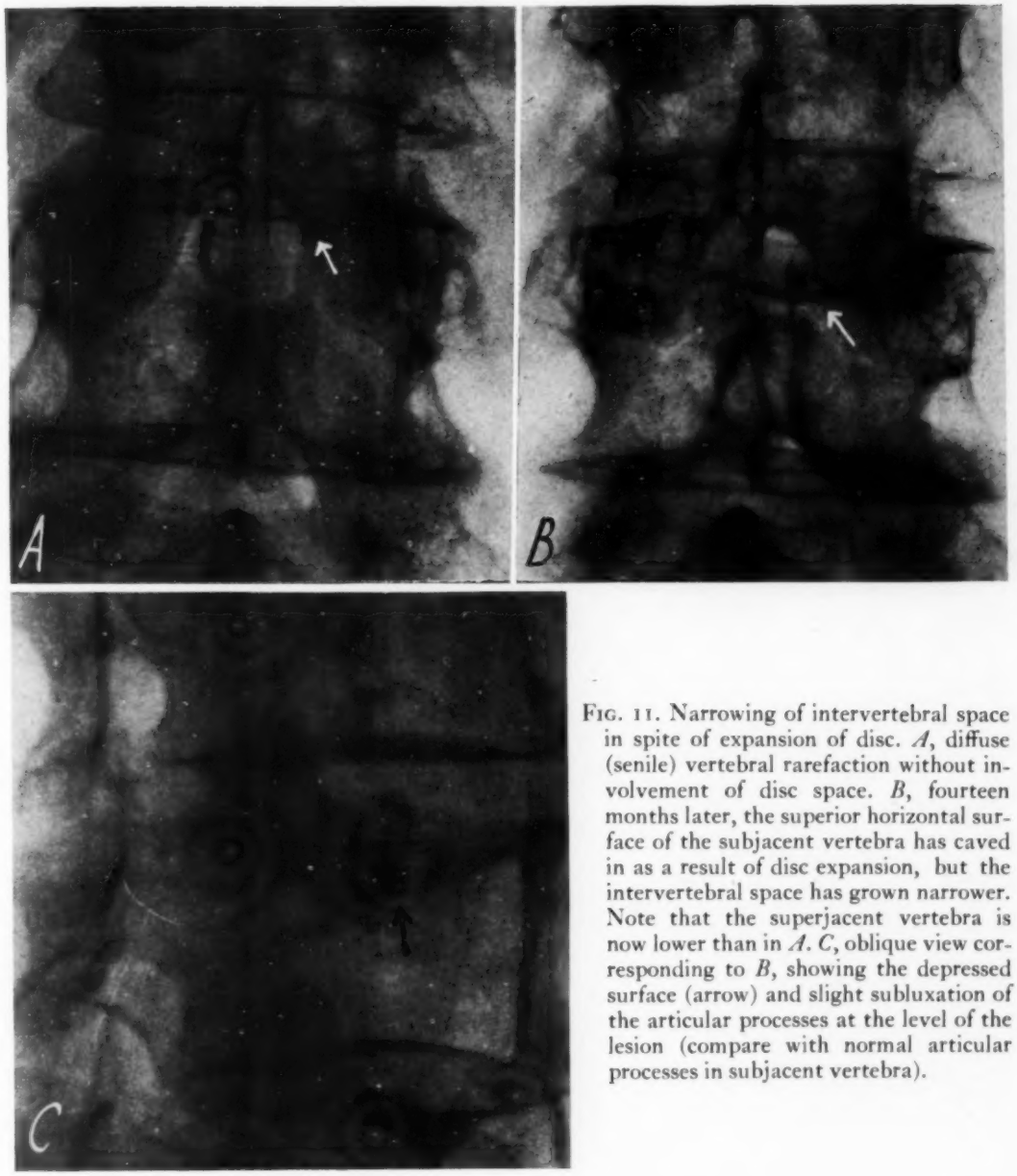

Fig. 11. Narrowing of intervertebral space in spite of expansion of disc. *A*, diffuse (senile) vertebral rarefaction without involvement of disc space. *B*, fourteen months later, the superior horizontal surface of the subjacent vertebra has caved in as a result of disc expansion, but the intervertebral space has grown narrower. Note that the superjacent vertebra is now lower than in *A*. *C*, oblique view corresponding to *B*, showing the depressed surface (arrow) and slight subluxation of the articular processes at the level of the lesion (compare with normal articular processes in subjacent vertebra).

mentation of the nucleus pulposus is not demonstrable on roentgenograms and is regarded as an incidental change.

4. DISEASES OF VERTEBRAL SYNOVIAL JOINTS

The apophyseal ("small," "posterior") joints, which connect the facets of contiguous articular processes, are synovial joints like the peripheral joints. Their lesions conform to the definition of arthritis[10] and may be classified as follows:

3. Diseases of vertebral synovial joints
   a. Acute arthritis
   b. Rheumatoid (atrophic) arthritis
   c. Osteo-arthritis (hypertrophic arthritis, degenerative joint disease)
   d. Secondary involvement

a. Acute Arthritis. This is marked by transient swelling of the apophyseal joint capsule.[10] In some instances the subchondral areas of the involved articular processes undergo slight rarefaction.[10,11] Together

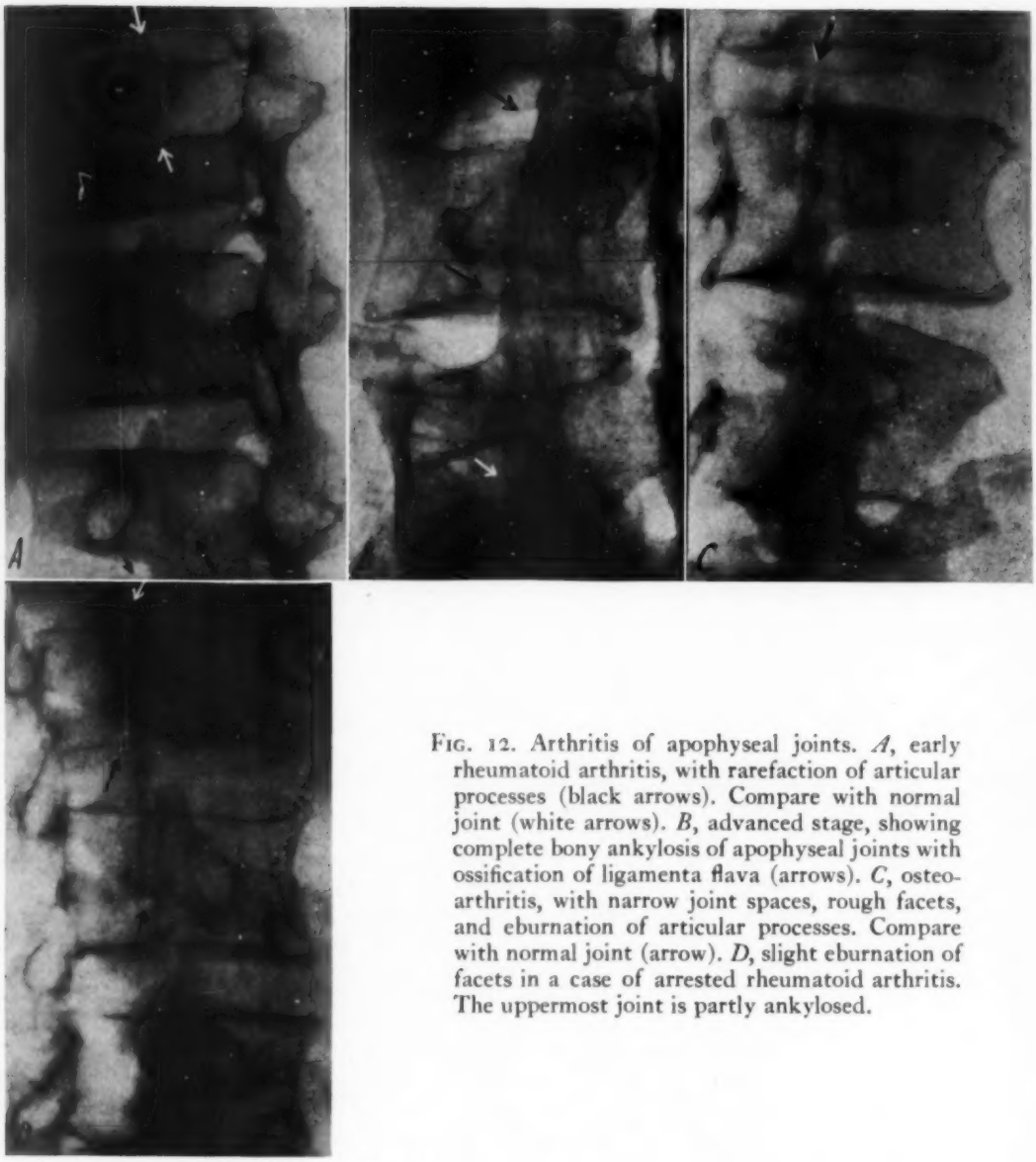

Fig. 12. Arthritis of apophyseal joints. *A*, early rheumatoid arthritis, with rarefaction of articular processes (black arrows). Compare with normal joint (white arrows). *B*, advanced stage, showing complete bony ankylosis of apophyseal joints with ossification of ligamenta flava (arrows). *C*, osteoarthritis, with narrow joint spaces, rough facets, and eburnation of articular processes. Compare with normal joint (arrow). *D*, slight eburnation of facets in a case of arrested rheumatoid arthritis. The uppermost joint is partly ankylosed.

with the occasional development of rheumatoid arthritis subsequent to acute arthritis, this is somewhat suggestive of an interrelation between the acute and rheumatoid types.[10] Acute spondylarthritis, which is usually confined to one or two apophyseal joints, subsides after a number of weeks in most cases, and the involved joint spaces and articular processes resume normal roentgenologic appearances.

b. Rheumatoid Arthritis. Rheumatoid arthritis of the apophyseal joints has the same histopathologic features as peripheral rheumatoid arthritis.[14] Roentgenologically, rarefaction of articular processes with narrowing of the intervening joint space (Fig. 12*A*) is noted at early stages.[10,11,14] The disease may evolve rapidly towards complete[21] or circling[14] bony ankylosis of the involved apophyseal joints; or it may become arrested before ankylosis has taken place,[14] in which case secondary reactions—such as roughening and condensation of the eroded facets[14]—may supervene (Fig. 12*D* and 13). As a rule, rheumatoid spondylarthritis involves a number of apophyseal

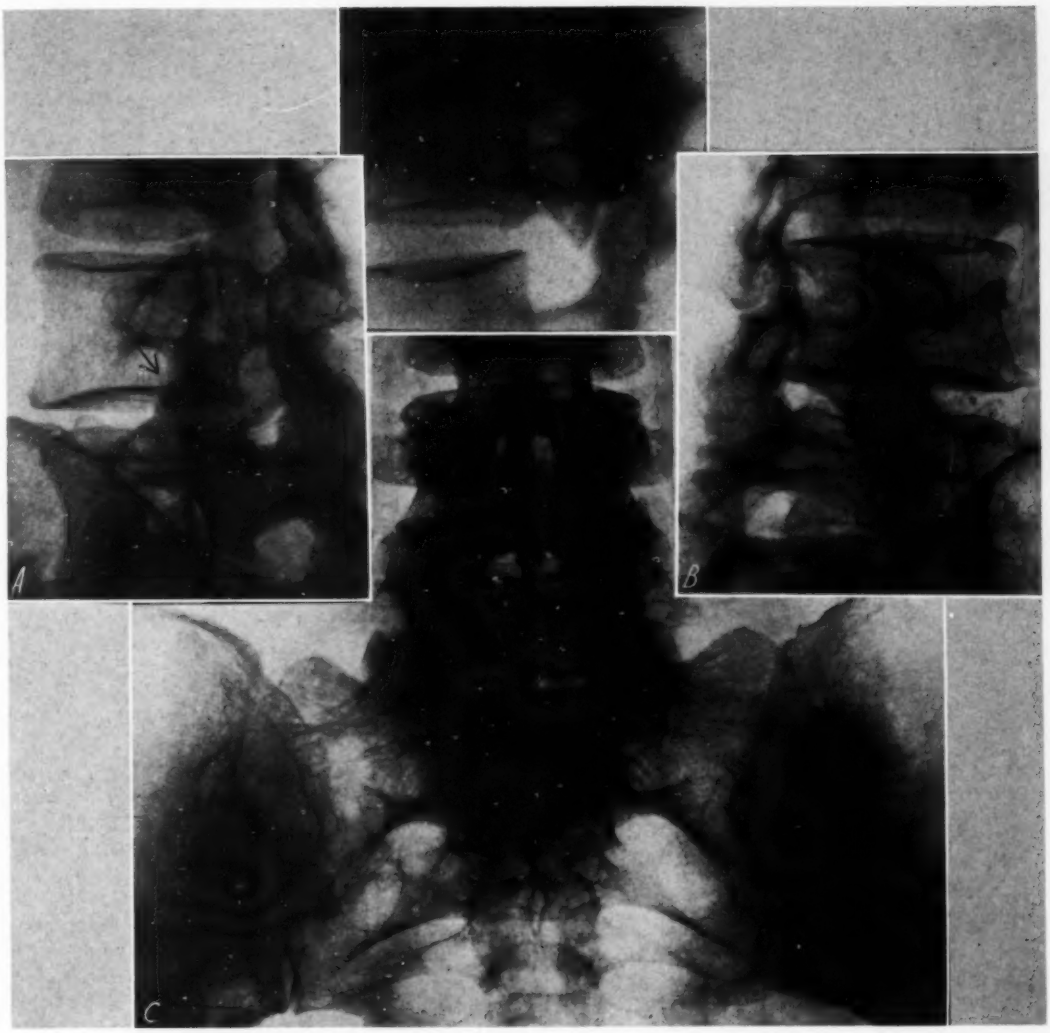

Fig. 13. Rheumatoid arthritis, confined to sacroiliacs and a pair of apophyseal joints in one single vertebral segment. *A*, fourth lumbar segment, right side, showing almost complete ankylosis of the joint (arrow). Compare with adjacent joints. *B*, same segment, left side, showing slight narrowing of joint with thorn-shaped osteophyte at tip of articular process, as a result of secondary osteo-arthritic reactions. *C*, sagittal view, showing involvement of sacroiliacs. Although only one pair of joints was involved, the patient was incapacitated with pain and complete rigidity of spine.

joints either simultaneously or successively and may finally affect the entire vertebral column; peripheral joints may then become ankylosed (Strümpell-Marie's disease); but the lesion may also remain confined to one single apophyseal joint,[14] or to a pair of joints in one vertebral segment (Fig. 13). The ligamenta flava, which merge with the joint capsules, may become ossified together with the joint capsules during the ankylosing phase of the disease (Fig. 12*B*). In about one-third of all cases, the longitudinal ligaments also become ossified at advanced stages[7,13,14,21]—hence the name, spondylitis ossificans ligamentosa.[7] Rarefaction of vertebral bodies is often noted during active phases of the disease and seems to correspond to the para-articular rarefaction of peripheral bones observed in the presence of peripheral rheumatoid arthritis; as the apophyseal arthritis progresses and becomes less active, the vertebral bone usually regains normal density.[11]

c. Osteo-arthritis. The anatomic findings

in osteo-arthritis of apophyseal joints do not differ from those of osteo-arthritis of peripheral joints.[18,21,22] Eburnation of the facets, osteophytes at the tips of the articular processes, and narrowing of the apophyseal joint space are seen on roentgenograms (Fig. 12*C*). These changes may develop during quiescent and arrested stages of rheumatoid arthritis, as already mentioned (Fig. 12*D* and 13*B*); this corresponds to the secondary osteo-arthritic reactions seen in the presence of arrested rheumatoid arthritis of peripheral joints. In most cases, however, osteo-arthritis of the apophyseal joints seems to be a consequence of subluxation of the articular processes due to some postural or static abnormality, such as is produced by thinning of the disc, scoliosis, fractures, and the like.[10]

d. Secondary Involvement. Infection may spread from the vertebral body into the neural arch, in the articular processes, and finally into the apophyseal joint (Fig. 14 and 1*C*). This is rare.[10,11]

Comments. Gouty, suppurative, and neurotrophic (Charcot type) arthritis of the apophyseal joints does not seem to have been recorded. Tuberculous arthritis, which sometimes involves the cervical apophyseal joints,[7] is considered here a secondary invasion in the presence of vertebral bone tuberculosis.

5. Diseases of Vertebral Ligaments

Not much has been written about the pathology of ligaments in general. The vertebral ligaments may be torn, and detached from the bone, as a result of trauma. Necrosis of vertebral ligaments may occur in the presence of vertebral bone necrosis, such as

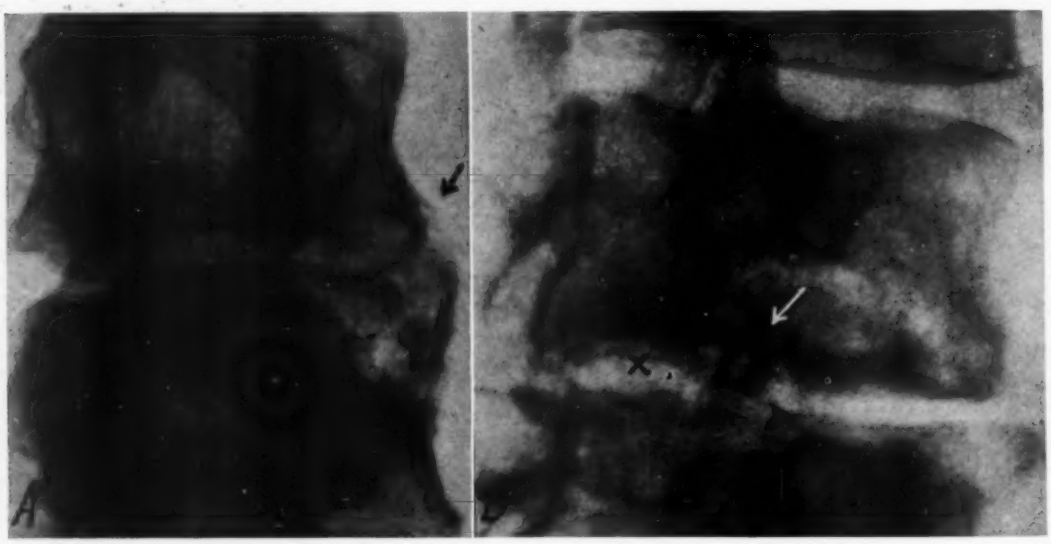

Fig. 14. Secondary involvement of apophyseal joint by pyogenic infection of vertebral body. The vertebral bodies show irregular bone structure with scattered areas of necrosis. One articular process is destroyed (*x*); the contiguous articular process is eroded (arrow).

due to tuberculosis,[23] osteomyelitis,[25] and syphilis.[7] These lesions of ligaments cannot be recognized on roentgenograms.

Ossification and Calcification. The ligaments calcify or ossify in the spine more frequently and more extensively than in most other parts of the skeleton. Ossification or calcification may occur at healing stages of vertebral bone infections, after fractures of the vertebrae, at advanced stages of rheumatoid spondylarthritis, and in old age (Fig. 4 and 15).

As a general rule, the vertebral ligaments become calcified as a result of disuse. For instance, when the mobility of vertebral regions is permanently diminished owing to

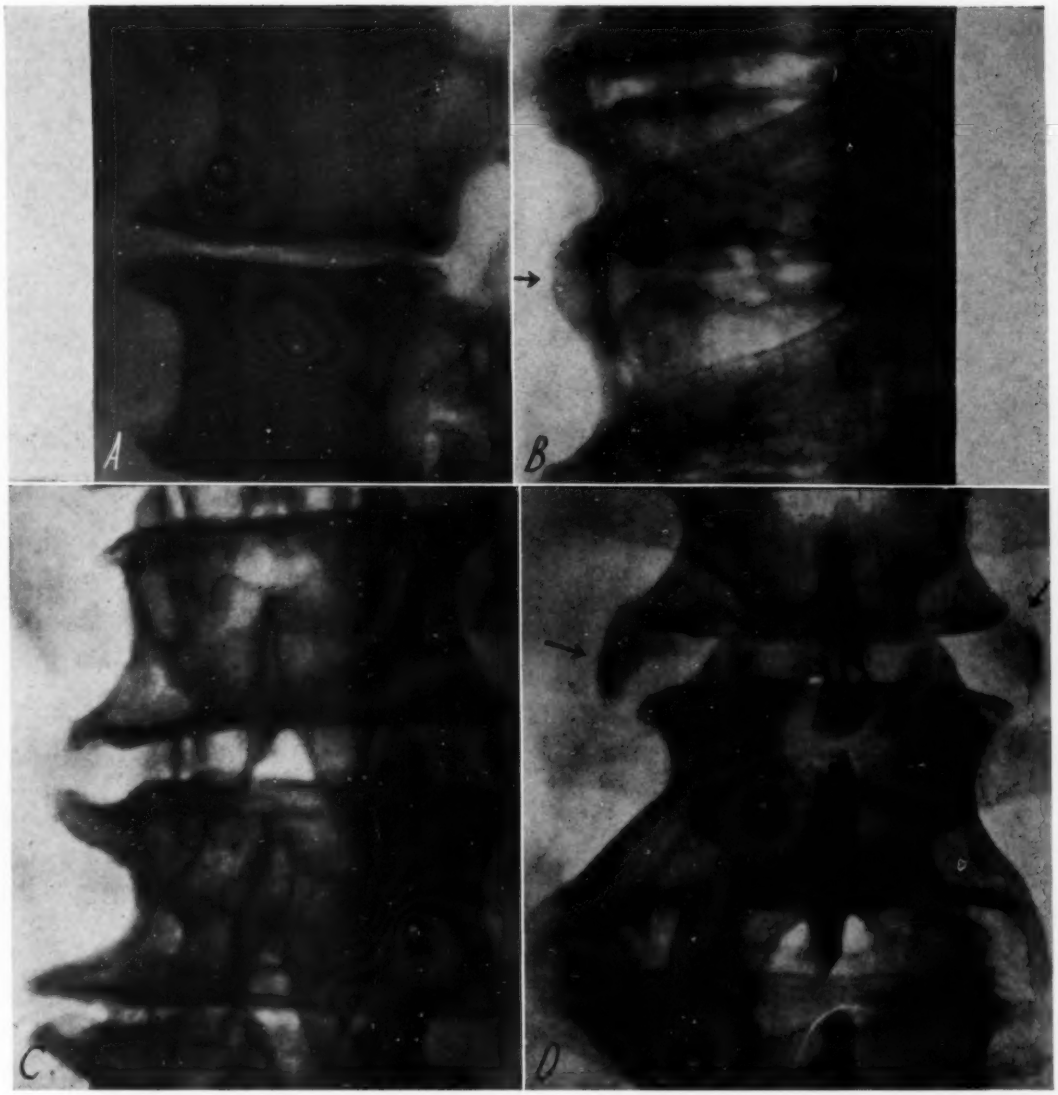

Fig. 15. *A* and *C*, marginal osteophytes at the level of thinned discs. *B* and *D*, ossified ligaments. Note that the osteophytes follow the course of the ligaments at the vertebral edges (see text).

disease or old age, the ligaments undergo calcification. This is nearly always more extensive in the thoracic spine, which has only scant mobility even normally, than in the more movable cervical and lumbar segments.[13] Calcification also occurs often in ligaments which have become relatively too long for the space which they bridge because this space has grown narrow—e.g. at the concave side of scoliotic and of kyphotic regions, and at the level of a narrowed disc space. Apparently, diminished tension is equivalent to disuse of ligaments.

Ossification of vertebral ligaments in the presence of Strümpell-Marie's disease is always associated with ankylosis of the apophyseal joints and occurs in only about one-third of these cases[14]; hence, it is considered here a secondary phenomenon.

Lesions originating in, and confined to, the vertebral ligaments do not seem to be on record. Morbid changes in the vertebral ligaments appear to be usually the result of disease of some other parts of the vertebrae

without being characteristic of any particular disease or group of diseases.[13]

6. CORRELATIONS WITH CLINICAL FINDINGS

Since this paper is concerned with only the roentgenologic aspects of vertebral disease, the discussion is confined to those correlations between clinical and roentgen findings which are significant for roentgen interpretation.

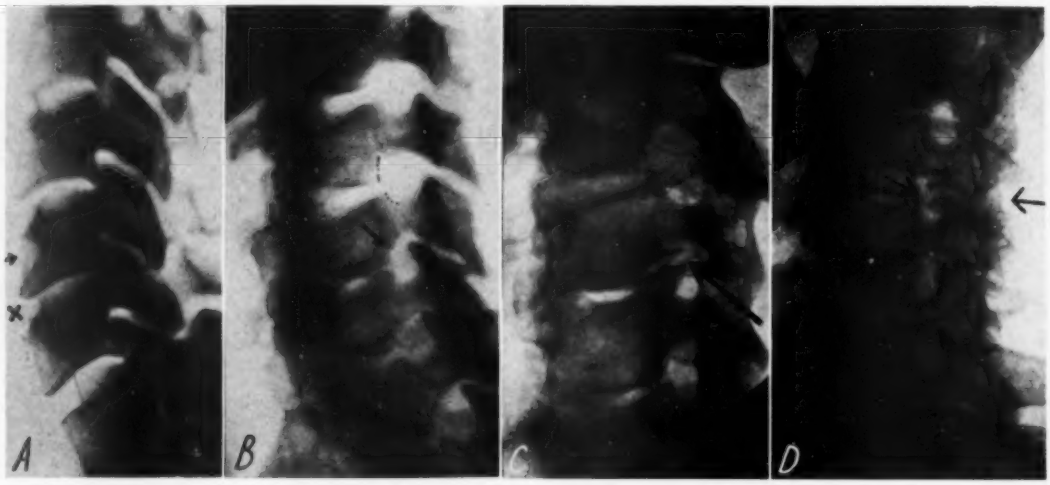

Fig. 16. Narrowing of neural foramina. *A* and *B*, the fifth cervical disc is flattened (*x*). *B*, oblique view to show the displacement of articular processes and narrowing of neural foramen at the level of the flat disc. *C*, osteophytes arising from posterior margins of vertebral bodies, and *D*, from tips of articular processes, encroach upon neural foramina.

a. Involvement of the Spinal Cord and Its Roots. Pain in the periphery, sometimes associated with trophic disturbances of the skin, muscles, and bones of the painful region,[12] may be caused either by compression of the spinal cord within the spinal canal, or by compression of nerve roots in the neural foramina.

Rupture of the disc into the spinal canal has come to be considered the most common cause of compression of the spinal cord, although anatomical and roentgenologic studies[6] have shown that clinical manifestations suggestive of disc rupture may also be the result of compression of the cord by osteophytes arising from the posterior surfaces of vertebral bodies,[17] by intraspinal protrusion of a "degenerated" but not ruptured disc,[6] by thickened and by ossified ligaments,[20] and, of course, by abscess and tumor.[23] Moreover, the clinical manifestations of rupture of the disc into the spinal canal can rarely be distinguished from those caused by compression of nerve roots in the neural foramina.[15] Finally, since in most cases a ruptured disc becomes flat, the neural foramina at this level become narrow, and the symptoms may then be due, at least in part, to compression of nerve roots in the neural foramina rather than to compression of the cord in the spinal canal. In the cervical and lumbar segments the nerve roots are thick and fill almost the entire neural foramen[5]; the distance between nerve root and surrounding bone is much smaller than the distance between spinal cord and surrounding bone. Consequently one may say that radicular manifestations are produced by compression of nerve roots more readily than by compression of the spinal cord.

Narrowing of neural foramina may be caused by different vertebral lesions, among which flattening of the disc is perhaps the most common.[15] Osteophytes arising from the posterior borders of the vertebral bodies[3,9] and from the articular processes

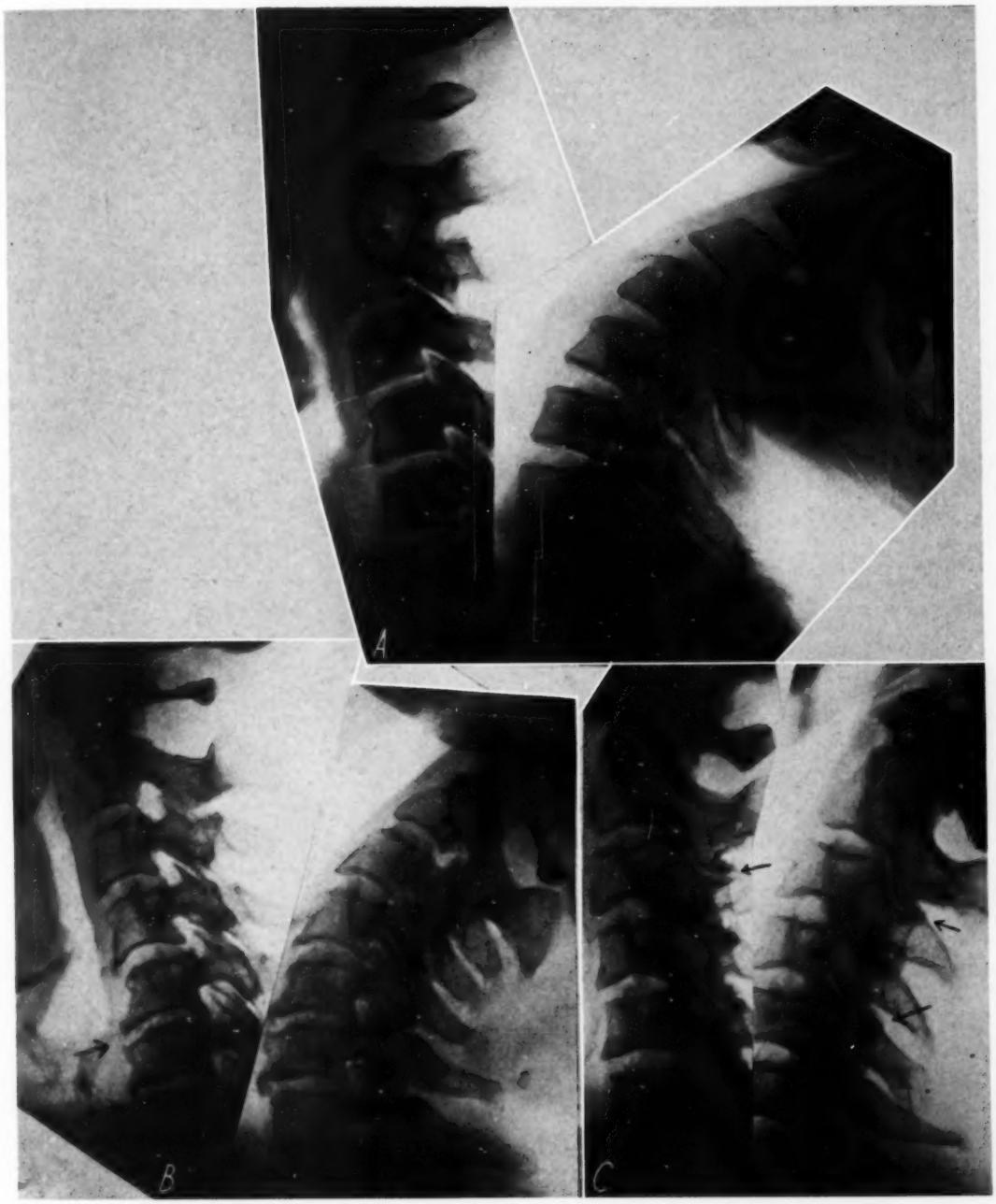

Fig. 17. Range of flexion and extension in cervical spine. *A*, normal. *B*, range of motion almost normal in spite of flattening of discs (arrows) and marginal osteophytes; apophyseal joints not involved. *C*, definite limitation of motion in the presence of normal discs but with apophyseal joints involved by osteo-arthritis (arrows). See text.

(Fig. 16); thickening and calcification of the apophyseal joint capsule[10] and of ligaments[10,20]; and subluxation of the articular processes[5] may also produce, or contribute to, compression of nerve roots. According to anatomical[5] and clinical[12] observations, the changes in and around the neural foramina disclosed on roentgenograms in these cases are actually responsible for the damage of the corresponding nerve roots.

Before considering surgical treatment for a lesion supposedly involving the spinal cord, one should make certain that the signs and symptoms are not produced by compression of nerve roots in the neural foramina. In this series clinical manifestations consistent with radicular neuritis were due to compression of nerve roots in 88 per cent of the cases, and to compression of the spinal cord in 12 per cent.

b. Limitation of Vertebral Movements. The mechanisms of vertebral motion are complex and variable. Extravertebral factors—such as the size of the chest, the amount of adipose tissue, and the spatial relations between ribs and pelvis—play a more important part than one would expect. In the cervical region, where these extrinsic factors are relatively insignificant, the mechanics of the spine can be studied more accurately than in other segments.*

The range of motion in the cervical spine varies individually and diminishes slightly with advancing years, but remains nearly constant from the third to the fifth decade. About 35 per cent of the patients with lesions of the vertebral symphyses (especially thinning of cervical discs) have a moderately diminished range of motion as compared with healthy persons of the same age group (Fig. 17); but in the remaining 65 per cent the range of motion is within normal limits, no matter whether osteophytes are present or absent at the corresponding

* The findings relating to vertebral motion are reported separately.

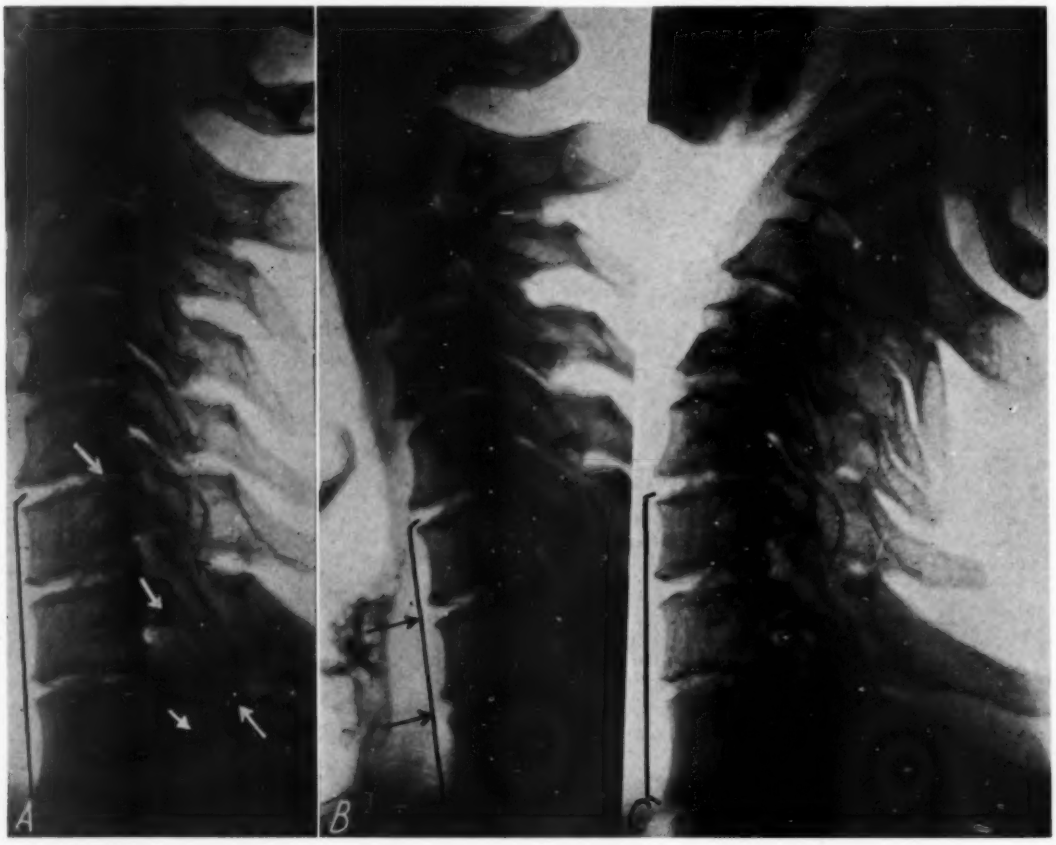

Fig. 18. *A*, all the cervical discs are flattened, but only the lower three apophyseal joints are involved. Note blurred outlines and narrowing of joint spaces (white arrows). *B*, forward flexion, and *C*, backward extension, showing movements practically normal in the segments with flat discs but normal apophyseal joints, while the vertebrae with involvement of apophyseal joints are almost immovable.

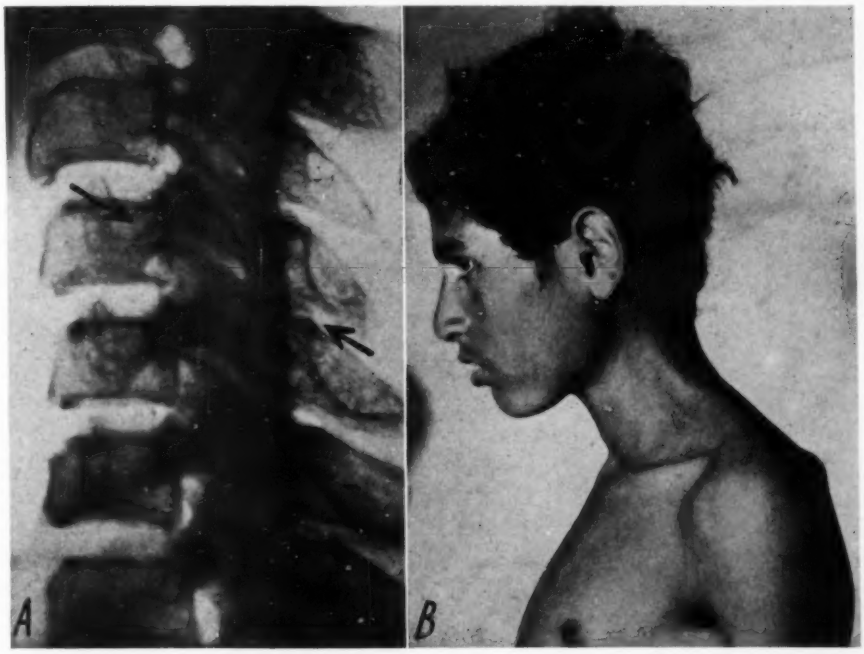

Fig. 19. *A*, roentgenogram of cervical spine, and *B*, photograph, of a sixteen year old Arab boy with rheumatoid arthritis of apophyseal joints, advanced stage. Complete immobility of neck. Only the upward motion of the eyeballs discloses the intention to stretch the neck backward. Note incomplete ankylosis of apophyseal joints with ossification of ligamenta flava. No trace of ossification of longitudinal ligaments; no involvement of discs and vertebral bodies. This shows the dominant part which the apophyseal joints play in the control of vertebral motion.

vertebral edges. When, however, the apophyseal joints have become involved secondarily, as a result of the "discogenetic" displacement of their facets, motion is definitely limited in most cases (Fig. 18). Restriction of movements, often amounting to immobility, is usually associated with lesions confined to, or originating in, the apophyseal joints (Fig. 17*C*, 18 and 19). As in peripheral joints, rheumatoid arthritis usually produces a greater limitation of movements than osteo-arthritis.

It has also been shown that pain in the involved vertebral regions is almost invariably present at active stages of the various types of apophyseal arthritis.[10,14] The observations indicate that lesions of the apophyseal joints conform to arthritis anatomically and roentgenologically, and that they produce the cardinal clinical manifestations of arthritis, viz., pain and limitation of movements. Thus there is no significant difference between arthritis of peripheral joints and arthritis of the vertebral synovial joints.

7. Conclusions

The main object of this study was to try and assign to the different vertebral diseases those places which they should occupy in the pathology of bone and joints, instead of regarding them separately as distinct entities. It was hoped that with this method of approach a better understanding of the pathology of the spine might be achieved. The findings indicate that differences which exist between bone and joint diseases and the same diseases involving the vertebral column are due merely to the particular anatomical structure and static function of the spine.

The various diseases of bone reappear in the vertebrae. Owing to its cancellous structure and abundant blood supply, the vertebral body is involved more frequently than the neural arch. Because of their frailty

relative to the weight they bear, the vertebral bodies undergo collapse and compression in the presence of rarefaction and necrosis more readily than other bones. The absence of connective tissue around the vertebral edge is responsible for the high incidence of marginal overgrowth. The clinical manifestations of vertebral bone disease conform to those of disease of other bones; but signs and symptoms peculiar to vertebral disease may become superadded when the vertebral symphyses become involved.

Lesions of the vertebral symphyses may have their origin either in the bone or in the disc. Bone disease close to, or at, the horizontal vertebral surface usually leads to involvement of the disc, mainly by interference with disc nutrition, as it seems. The disc itself may become diseased in the absence of a bone lesion. In both cases, the disc grows flat, and the distance between the vertebrae diminishes proportionally. This, in itself, does not produce clinical manifestations; it causes neither pain nor limitation of movements. When, however, the apophyseal joints have become involved as a result of the "discogenetic" displacement, pain in the involved region, together with limitation of movements, is apt to appear. At the same time, but independently, narrowing of the neural foramen may lead to compression of nerve roots, provided the root is thick and the narrowing sufficiently marked; and manifestations of radicular neuritis develop. As a rule, compression of nerve roots has a much higher incidence than involvement of the apophyseal joints in the presence of lesions of the symphyses; hence the conspicuous predominance of symptoms felt in the periphery over symptoms felt in the spine in this group of cases.[15] When neither the nerve roots nor the apophyseal joints are affected, lesions of the vertebral symphyses usually remain silent, unless there is a herniation of the disc into the spinal canal.

The various forms and phases of arthritis occur in the apophyseal joints and are associated with the main clinical manifestations of arthritis, viz., pain in the involved region and limitation of its movements. The range of motion of the spine is controlled by the apophyseal joints, but not by the symphyses. Evidence of nerve root involvement may be noted when the nerve roots become compressed by the inflammatory, fibrous, or bony changes caused by the arthritic process.

Of the lesions of the vertebral ligaments, only calcification and ossification are disclosed roentgenologically. These are common reactions, not characteristic of any particular disease, and are apparently the result mainly of disuse of ligaments. They do not seem to produce clinical manifestations; but the signs and symptoms of the vertebral disease which has caused calcification or ossification may be present.

It would seem that the clinical manifestations of vertebral diseases depend not so much on the nature of the lesion as on the involvement of the different parts of the vertebrae and on the level of the morbid process. For example, when bone disease involves the apophyseal joints, the manifestations of arthritis become added to those of osteitis, and so forth, Again, as regards the level of the lesion, narrowing of neural foramina is clinically significant mainly in the cervical and lumbar regions, where the nerve roots are thick, but often fails to cause symptoms when the thoracic spine, with its thin nerve roots, is involved. Similarly, apophyseal arthritis is less disabling in the thoracic spine, which has scant mobility, than in the more movable cervical and lumbar regions. Factors of this kind should be taken into account when evaluating the clinical significance of the roentgen findings; for they would seem to explain, at least in some measure, the discrepancy between the incidence of anatomical lesions and the incidence of clinical manifestations in disease of the spine.

8. SUMMARY

1. The diseases of the spine are regarded as vertebral localizations of bone and joint

disease and are classified as (1) diseases of vertebral bone (spondylosteitis); (2) diseases of vertebral symphyses; (3) diseases of vertebral synovial joints (spondylarthritis); (4) diseases of vertebral ligaments.

2. In general, the morphologic appearances and clinical manifestations of a given vertebral disease correspond to those of the same disease involving other bones and joints. Accordingly, there does not seem to be a definite need for a separate classification of vertebral diseases.

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THE IMPORTANCE OF EXAMINATION OF THE SPUTUM IN EVALUATING PARENCHYMAL INFILTRATION*

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SPIVAK, COLORADO

IN AN article[1] in a recent issue of the JOURNAL, I presented certain specific cases where the diagnosis of pulmonary tuberculosis was made on roentgen changes *per se* which subsequently proved to be non-tuberculous in origin and character.

The late Lawrason Brown wisely stated that "tuberculosis is seen and not heard." The 1940 edition of Diagnostic Standards of the National Tuberculosis Association[2] states, "the x-ray is the foundation of the early diagnosis of pulmonary tuberculosis." The roentgenogram usually depicts changes which may be considered with some degree of certainty as being due to pulmonary tuberculosis. The early infiltrate, the exudative process, the exudative-productive, caseous pneumonic, ulcerative, productive, fibrotic and calcareous lesion may be the photographic expression of the pathologico-anatomical tuberculous pulmonary change. However, identical roentgen findings may be seen in pathologic entities not due to pulmonary tuberculosis. Because of the similarity of roentgen findings in tuberculous and non-tuberculous pulmonary conditions, roentgen changes produced by tuberculosis of the lungs cannot be considered pathognomonic for that disease.

There is a symptom complex that is usually associated with active pulmonary tuberculosis. Nevertheless, the dry or productive cough, the streaked sputum, hemoptysis, fever, pleurisy, loss of weight, malaise and associated symptoms are often seen in patients with abnormal roentgen findings not due to pulmonary tuberculosis. Still, the occurrence of these symptoms, singly or in combination, particularly in the presence of abnormal roentgen findings, immediately leads to the diagnosis of pulmonary tuberculosis, even though tubercle bacilli have never been found in the sputum.

The 1940 edition of Diagnostic Standards states that in a patient "with a demonstrable parenchymal infiltration in the lung that is apparently active, when tubercle bacilli cannot be demonstrated, there is probably a non-tuberculous lesion." In the presence of open pulmonary tuberculosis or active ulcerative endobronchial tuberculosis, tubercle bacilli are found in the sputum by the usual methods of identification. Where there is a failure to demonstrate tubercle bacilli at any time in the sputum originating from below the bifurcation of the trachea, the phthisiologist should immediately consider the probable presence of a non-tuberculous pulmonary condition, regardless of suggestive tuberculous roentgen changes.[1]

DISCUSSION

Table 1 represents an analytical study of 18 cases which were referred to the Sanatorium of the Jewish Consumptives' Relief Society at Spivak, Colorado, with the preadmission diagnosis of pulmonary tuberculosis in various stages of the disease. A number of these patients had symptoms that could easily be construed as being due to active pulmonary tuberculosis. Of the 18 patients, 14 had a productive cough varying from slight to severe in intensity. Symptoms of bronchial irritation were entirely absent in the patients with sarcoma of the mediastinum, aleukemic lymphatic leukemia with lung infiltrations and Boeck's sarcoid, respectively. Subfebrile

[1] Rest, A. Roentgenogram and some chronic non-tuberculous pulmonary conditions. AM. J. ROENTGENOL. & RAD. THERAPY, 1944, *51*, 434–438.
[2] Diagnostic Standards of National Tuberculosis Association. 1940.

* From the Medical Department of the Sanatorium of the Jewish Consumptives' Relief Society, Spivak, Colo.

Table I

ANALYSIS OF EIGHTEEN PATIENTS WITH NON-TUBERCULOUS PULMONARY CONDITIONS DIAGNOSED AS TUBERCULOSIS

Case No. and Sex	Age	Pre-admission Diagnosis	Symptoms upon Admission	Roentgen Findings	Sputum	Subsequent Diagnosis
1 M	29	F.A.B. Miliary T_3 R_3 L_3	Marked fatigue, dyspnea	Extensive bilateral infiltrations	Negative	Aleukemic lymphatic leukemia*
2 F	31	Min. A. T_1 R_1 L_0	Moderate cough and expectoration; subfebrile temperature	Small mottling off rt. hilum; linear densities along 1st and 2nd interspace trunks	Negative	Hodgkin's disease
3 F	33	F.A.A. T_3 R_3 L_3	Productive cough with streaked sputum; dyspnea; left-sided pleurisy	Generalized bilateral mottling; enlargement of heart	Negative	Mitral stenosis and insufficiency
4 M	56	F.A.A. T_3 R_2 L_2	Severe cough and expectoration; repeated hemoptyses; subfebrile temperature	Hazy dense shadows in both bases	Negative	Bilateral non-tuberculous bronchiectasis
5 M	43	F.A.A. T_3 R_0 L_3	Slight productive cough; subfeb. temp.; streaking; chills and night sweats; left-sided pleuritic pain	Irregular rounded opaque shadow in left upper lobe	Negative	Sarcoma of mediastinum
6 M	23	Mod. A.B. T_1 R_0 L_2	Left-sided pleurisy; moder. productive cough; subfebrile temperature	Opaque mass in left upper lobe originating in mediastinum and extending to nipple line	Negative	Sarcoma of mediastinum with metastases
7 M	30	F.A.A. T_3 R_3 L_3	Severe cough and expectoration; bilateral pleuritic pain	Extensive infiltration in both lungs with exudation; annular shadow in left upper lobe	Negative	Boeck's sarcoid*
8 M	21	F.A.A. T_3 R_3 L_1	Asymptomatic	Numerous productive nodules in both lung fields, particularly in both midlung fields	Negative	Boeck's sarcoid
9 F	59	F.A.B. T_3 R_3 L_3	Productive cough; hemoptyses; subfebrile temperature	Infiltrations in major portion of right lung and left upper lobe with annular shadow in left upper lobe	Negative	Hypernephroma* of left kidney with metastases to both lungs and liver; diabetes mellitus; malignant hypertension
10 M	63	F.A.B. T_3 R_3 L_3	Marked productive cough; dyspnea, hemoptyses	Probable cavity off left hilum; coarse infiltrations in both bases	Negative	Bilateral non-tuberculous bronchiectasis
11 F	54	F.A.A. T_3 R_3 L_3	Severe productive cough	Scattered areas of infiltration in both lungs	Negative	Bilateral non-tuberculous bronchiectasis
12 M	52	Mod. A.B. T_1 R_0 L_2	Severe productive cough	Dense shadow extending outward from hilum to the chest wall	Negative	Carcinoma of lung*
13 F	69	F.A.A. T_3 R_3 L_3	Slight cough and expectoration; slight dyspnea; loss of weight	Dense shadow off right hilum; coarse nodules throughout both lungs	Negative	Carcinoma of lung
14 M	56	Mod. A.A. T_2 R_2 L_0	Severe cough and expectoration; subfebrile temp.; malaise, loss of weight	Opaque shadow in right upper lobe	Negative	Carcinoma of lung
15 M	65	Mod. A.B. T_2 R_0 L_2	Severe productive cough; subfebrile temperature; dyspnea; loss of weight	Opaque wedge-shaped shadow off left hilum	Negative	Carcinoma of lung
16 M	60	Mod. A.A. T_2 R_2 L_0	Slight productive cough; subfeb. temp.; extreme weakness; loss of weight; dyspnea	Large oval shaped opaque shadow in right upper lobe	Negative	Carcinoma of lung; hyperthyroidism
17 M	26	F.A.A. T_3 R_3 L_3	Slight productive cough	Massive bilateral fine infiltrations apex to base	Negative	Calcicocis of lungs*
18 M	42	F.A.A. T_3 R_3 L_3	Severe productive cough, marked dyspnea, cyanosis	Scattered areas of infiltration and fibrosis with cavitation right upper lobe; fibrosis left upper lobe	Negative	Ayerza's disease

* These cases form the basis for the previous article.[1]

The classifications used were those of the National Tuberculosis Association as described in the Diagnostic Standards of the National Tuberculosis Association[2] which divides tuberculous lesions into minimal, moderately advanced and far advanced, respectively, depending on degree of involvement. The minimal lesion involves one-third of the volume of one lung without cavitation, the moderately advanced is more extensive than one-third the volume of one lung and not greater than the volume of one lung with cavitation not larger than one interspace. The far advanced lesion is more extensive than the volume of one lung with cavitation larger than one interspace.

The Turban-Gerhardt classification which was used designates the anatomical distribution of the lesion. For descriptive purposes, T_1 R_1 L_0 signifies a minimal lesion of the right apex with no involvement of the left lung; T_2 R_2 L_0, a moderately advanced lesion of the right lung with no involvement of the left lung; and T_3 R_3 L_3, a far advanced lesion of both lungs.

temperature, dyspnea, streaking, hemoptysis, pleurisy and malaise characterized the symptoms of a large number of the cases studied. The roentgenogram showed various changes sufficient to classify the patients from moderately to far advanced, with the exception of the case of Hodgkin's disease, where only minimal changes existed. There were roentgen manifestations that gave the appearance of miliary infiltrations, acinose and acinose-nodose nodules, the dense opacity of extreme fibrosis with localized pachypleuritis, exudative processes and cavitation. Because of the pulmonary and constitutional symptoms and the abnormal roentgen findings, these patients were diagnosed as having pulmonary tuberculosis and were admitted to our sanatorium for treatment. The principal diagnostic feature, namely positive sputum, was absent in all these 18 cases, and because of this finding alone, we were of the opinion that pulmonary tuberculosis did not exist in these patients.

In my previous article[1] I emphasized to the roentgenologist the fact that other lung conditions can produce roentgen changes which can easily be interpreted as due to pulmonary tuberculosis. This presentation is intended to stress the importance of demonstrating the tubercle bacillus in sputum in order to properly evaluate roentgen changes as being due to tuberculosis.

SUMMARY

Eighteen cases are presented which had roentgen changes simulating pulmonary tuberculosis, and from these roentgen changes one was classified as minimal, five as moderately advanced, and twelve as far advanced prior to admission to our sanatorium. The roentgen findings were diverse and ranged from minimal to extensive lung changes. Only one was asymptomatic, while all the others had symptoms that varied from marked dyspnea and fatigue to severe productive cough, subfebrile temperature, loss of weight and hemoptysis. However, none of them ever had sputum positive for tubercle bacilli.

Subsequent examinations revealed that four had primary carcinoma of the lung, two had Boeck's sarcoid, two had sarcoma of the mediastinum, three had non-tuberculous bronchiectasis, while the others had aleukemic lymphatic leukemia with lung changes, Hodgkin's disease, mitral stenosis and insufficiency, calcicosis of the lungs and Ayerza's disease respectively.

CONCLUSIONS

1. It is well understood that certain non-tuberculous pulmonary conditions may produce roentgen changes in the lungs that are indistinguishable from those produced by tuberculosis.

2. These non-tuberculous lung conditions may even produce clinical symptoms that simulate very closely, if not exactly, the symptoms produced by tuberculosis.

3. The absolute diagnostic feature is the demonstration of the tubercle bacillus in the sputum. We have found axiomatically that when a patient expectorates sputum from below the bifurcation of the trachea, the sputum contains tubercle bacilli if an open lesion of tuberculosis exists. If the sputum has always been negative, search should be made for a non-tuberculous pulmonary condition, regardless of how characteristic the roentgen changes are for tuberculosis.

4. The roentgenologist's diagnosis of the chest roentgenogram for tuberculosis should be presumptive rather than absolute, unless the clinical data reveal that the sputum is positive for tubercle bacilli.

5. Adenocarcinoma of the lung is relatively slow growing and in the early stages may produce very slight pulmonary symptoms and, as shown in my study, may easily be mistaken for inactive or mildly active pulmonary tuberculosis. The presence of a parenchymal lesion in individuals in the fifth or sixth decade who have persistently negative sputum examinations should lead to the investigation for carcinoma, the adeno form of which is apparently responsive to early radical surgery.

Vol. 53, No. 4

HEMANGIO-ENDOTHELIOMA OF THE DIAPHRAGM

REPORT OF A FATAL CASE IN AN INFANT

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EWING'S classical description of vascular tumors, particularly the endothelioma group,[1] shows the great difficulty in classifying these tumors. They are rather common in the skin as telangiectasias and are occasionally hereditary (Rendu-Osler-Weber's disease). Occasionally they are seen in other organs, often as incidental findings. They are usually benign. Their occurrence in infancy is rather rare.

Döderlein[2] reports a similar case occurring in an infant. His case is comparable to this one in that they both occurred in the right leaf of the diaphragm and were apparently the cause of death.

H. G.,* infant baby girl, was born September 13, 1942, at 5:30 A.M. in the Maternity Division of the Binghamton City Hospital at the end of the seventh month of uneventful gestation. The infant was the first child of healthy white American parents. Delivery was normal and the child responded well and appeared perfectly normal. The present and past parental history was negative, especially in regard to telangiectasias and nevi. The birth weight was 5 lb. 10 oz. The infant was breast fed and appeared entirely normal until September 24, the eleventh day of life, at which time the mother noticed a slight grunting noise as the child nursed. The temperature and respirations were normal and no vomiting had occurred. However, the attending physician requested a roentgen examination for the presence of a possible enlargement of the thymus. The following were the roentgenoscopic and roentgenographic findings:

The thorax appears normal in contour. The superior mediastinum is not remarkable and thymic enlargement is apparently not present. The left diaphragm is normal in position and moves normally with respiration. Normal gas shadows are present below. The heart appears normal in configuration and the pulsations are not unusual. The lower liver margin is clearly defined and in normal position. The lung fields are clear and appear well aerated. The right diaphragm is obscured by a dense shadow triangular in shape with a smooth upper margin occupying the entire cardiophrenic angle from the outer third of the diaphragm to the base of the heart shadow. This shadow appears fixed to both diaphragm and right heart border and does not move on inspiration. In lateral projection the mass is in the anterior portion of the right thorax. The nature of this increased density is not known.

The temperature was not elevated, blood and urine examinations were normal.

On September 27, a definite cyanosis was present. Respirations were increased and there appeared to be some difficulty in swallowing. Vomiting was present and feeding was accomplished with great difficulty. Oxygen was occasionally administered as well as stimulants. A second roentgen examination was made with similar findings, except that both lungs appeared more hazy, possibly due to fluid in the pleural spaces. The course was progressively worse and the child died on the twenty-sixth day apparently of asphyxiation. The laboratory findings had all been negative with the exception of the roentgen findings. The cause of death was not determined ante mortem.

The roentgenologic consultation offered the following possibilities: (1) Middle lobe atelectasis. The shadow was in the anatomical position of the middle lobe but not typical of this condition. (2) Atelectasis of a supernumerary or infracardiac lobe. This could not be ruled out. (3) An inflammatory process or consolidation of lung tissue. This was possible but the absence of fever and normal blood findings were opposed to this diagnosis. (4) Congenital heart lesion. The absence of murmurs was thought evidence against this. (5) Diaphragmatic her-

[1] Ewing, J. Neoplastic Diseases. Fourth edition.

[2] Döderlein, H. Ueber ein selten ausgedehntes Hämangiom des Zwerchfells sowie der inneren Brust- und Bauchwand als Todesursache bei einem Neugeborenen. *Zentralbl. f. allg. Path. u. path. Anat.*, 1938, *71*, 193–201.

* This case is presented through the courtesy of Dr. H. I. Johnston of the Obstetrical Staff of the Binghamton City Hospital.

nia through the esophageal hiatus or through the parasternal portal. This was a definite possibility and a barium study was suggested but was not attempted because of the critical condition of the infant. (6) Eventration of the diaphragm. Shadows were not typical. (7) Her-

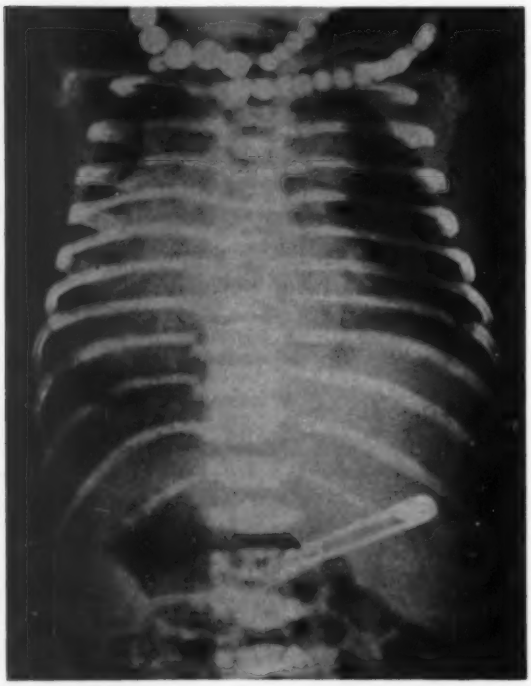

Fig. 1. Triangular shadow of homogeneous density in medial portion of the right lower lung field.

nia of the liver. The lower liver margin appeared normal. (8) Hernia of the pericardium, dermoid cyst and encysted fluid were considered but could not be proved. (9) A tumor of the pericardium, diaphragm or liver was also thought of, as was also the possibility of abnormally situated thymic tissue. The final diagnosis rested upon the postmortem findings.

The pathologist's report follows. Autopsy: H. G., baby girl, aged twenty-six days; American. The body was that of a well developed white female infant weighing 2,100 grams. It was extremely pale and was still warm.

A circular incision was made from shoulder to shoulder around the chest. Difficulty was experienced at once in attempting to remove the chest plate which was unusually adherent and attached to the diaphragm. When this was cut through, the diaphragm was found to measure up to 2 cm. in thickness reaching from the anterior attachment to the ribs and sternum to the posterior attachment of the diaphragm and laterally involving about two-thirds of the diaphragm (right leaf). The thickening extended up into the pericardium about two-thirds of which was involved and which appeared to be continuous with the thickening of the diaphragm. In the pericardium this thickening extended up to the reflection around the great vessels. The thymus which was not large could be identified and appeared to be separate from the pericardium from which it could be easily dissected. However, small masses of the tumor tissue appeared to extend into the substance of the thymus. On section, the thickened pericardium and diaphragm consisted of very dark red, very fine spongy tissue, from which some blood could be expressed, and the tissue was definitely resilient. The part of the pericardium

Fig. 2. Shadow appears in anterior portion of the pleural space.

not involved was on the left lateral portion and the heart present in the sac was apparently normal and posterior to the tumor mass. There were no pericardial adhesions. The aorta and the pulmonary artery were markedly compressed by the tumor. Both pleural cavities contained large amounts of yellow fluid which

compressed the lungs, producing extensive atelectasis. The examination of the diaphragm made it possible to inspect the abdominal viscera which were all normal.

Anatomical findings: (1) tumor of diaphragm and pericardium causing compression of the large vessels of the heart; (2) extensive large bilateral pleural effusion causing extensive compression of the lungs; (3) normal thymus, lungs and abdominal viscera.

Microscopical findings: Hemangio-endothelioma confined to the diaphragm and extending into the pericardium with considerable hemosiderin deposited with the sinuses; heart, thymus, adrenals, kidneys, spleen and liver normal; lungs—extreme atelectasis, passive congestion and deposition of hemosiderin.

The above case represents an interesting and extremely rare tumor.

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RESULTS OF IRRADIATION OF OVARIAN TUMORS*

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CARCINOMA of the ovary is one of the most fatal of pelvic neoplasms and treatment has always presented difficult problems. Treatment by surgery alone results in a low "cure rate." Our observations and those of others appear to indicate that considerable improvement of this cure rate can be expected by the addition of postoperative irradiation. An accurate comparison of results reported by different investigators is difficult, however, because of the differences of clinical and pathologic classification of these tumors, as well as the small number of cases which are usually reported.

TABLE I
AGE DISTRIBUTION

Years	
20–29	5
30–39	19
40–49	27
50–59	35
60–69	10
70–79	4
Total	100

This paper deals with the results of combined surgical and roentgen treatment of 100 consecutive patients diagnosed clinically or pathologically as having carcinoma of the ovary. They were seen at the University Hospitals from 1930 to 1938, inclusive. Ninety-nine of them were treated from 1930 through 1938, while one patient who was first treated in 1926 received repeated courses of treatment, the last in 1931. All patients were traced and those living have been observed from 60 to more than 155 months. The last communication from all but one of the living patients was received in January, 1944, and this patient was last heard from in November, 1942—more than five years after irradiation.

Age. The age distribution is shown in Table I and it can be seen that the majority of cases occurred in the fifth and sixth decades.

Classification. Most authors who have reported results of treatment of ovarian tumors have classified their cases in three or four clinical groups. Since the basis of the classification has been the operative findings, the groups are similar. The classification which we have used is essentially that of Heyman[5] who has divided his cases into four groups:

Group I: Those in which removal of the primary tumor and all visible metastases was possible.

Group II: Those who had (*a*) partial or total removal of the primary lesion but in whom there were visible metastases remaining; or (*b*) ascites with malignant cells or spill of malignant cells into the peritoneal cavity. (We have added "*b*" to Heyman's classification.)

Group III. Recurrence of malignant tumor following either operation or irradiation.

Group IV: Inoperable tumor; exploratory operation or paracentesis only; distant metastases.

The distribution of our cases in the four clinical groups is shown in Table II. Twenty-one patients were classified in Group I, 48 in Group II, 8 in Group III and 17 in Group IV. One patient was not classified as she received preoperative irradiation and died three months later of cardiac failure without being operated upon. Five patients had non-malignant tumors and were therefore not classified. These figures show that in 73 patients the tumor had progressed to a stage which required more than surgical removal. Ascites was present in 25 patients.

* Presented at the Joint Meeting of the American Roentgen Ray Society and the Radiological Society of North America, Chicago, Ill., Sept. 24–29, 1944.

Operation, either before or after irradiation, was performed on 94 patients. Four showed such extensive involvement that paracentesis alone was done. One patient had obvious metastases to the vagina and biopsy of only this lesion was taken while one patient was not operated upon.

Location. The tumor was unilateral in 46 cases (Table III). Bilateral involvement—either primary or secondary—was found in 30 cases. In 24 cases no information on this point was available. These included those operated upon elsewhere, those who were tapped only and those in whom the operative report failed to mention the site of the neoplasm.

TABLE II

CLINICAL GROUPS

Group I	21
Group II	48
Group III	8
Group IV	17
Unclassified	1*
Benign tumors and non-neoplastic disease	5
Total	100

* Patient was given preoperative roentgen therapy and died three months later of heart failure.

Pathology. Histopathologic diagnosis was obtained in 98 of the cases. Of the remaining 2, one could not undergo an operation because of heart failure, while the other patient was tapped repeatedly without finding malignant cells in the sediment. Physical examination, however, showed evidence of far advanced abdominal carcinomatosis and the patient died seven months later at home.

As Table IV indicates, there were 91 malignant and 2 potentially malignant neoplasms, 2 benign neoplasms and 3 non-neoplastic conditions. The 2 patients diagnosed as having papillary cystadenomas really had malignant neoplasms since one showed evidence of massive recurrence and died with ascites five years later, while the other showed metastases and adhesions at the time of operation and only a biopsy was taken. In the series with malignant neoplasms, 3 patients came to us with the report that a malignant cyst had been removed elsewhere. Papillary cystadenocarcinoma was the most common pathologic finding; the 47 cases make up nearly half of all the malignant neoplasms. The remainder of the malignant neoplasms is divided into 5 carcinomas (all of these cases were operated upon elsewhere but the reports coming from other laboratories could not be checked by us), 21 adenocarcinomas, 9 pseudomucinous cystadenocarcinomas, 1

TABLE III

LOCATION

Left	18
Right	28
Bilateral	30*
Not stated	24
Total	100

* One patient had a papillary cystadenocarcinoma on the left and a Brenner's tumor on the right.

TABLE IV

PATHOLOGIC DIAGNOSIS

A. *Potentially malignant*		
Granulosa cell tumor	1	
Theca cell tumor	1	2
B. *Malignant neoplasms*		
Carcinoma	5	
Adenocarcinoma	21	
Papillary cystadenoma	2*	
Malignant cyst	3	
Papillary serous cystadenocarcinoma	47	
Pseudomucinous cystadenocarcinoma	9	
Disgerminoma	1	
Teratoma with epidermoid carcinoma	1	
Malignant dermoid cyst	1	
Epidermoid carcinoma	1	91
C. *Benign neoplasms*		
Fibroma	2	2
D. *Non-neoplastic diseases*		
Simple serous cyst	1	
Tubo-ovarian abscess	1	
Ectopic pregnancy	1	3
E. *Miscellaneous*		
No biopsy	1	
No cancer found	1†	2
Total		100

* The clinical findings and subsequent course in both patients were those of malignant neoplasm.

† This patient was tapped repeatedly, but no neoplastic cells were found in the sediment. The clinical findings and course were those of advanced ovarian neoplasm.

disgerminoma, 1 teratoma with epidermoid carcinoma, 1 malignant dermoid cyst and 1 epidermoid carcinoma. This last diagnosis was made on a patient who came to us with recurrent carcinoma of the omentum and mesentery and who gave a history of having had a malignant ovarian tumor removed twelve years previously. The 2 benign neoplasms were fibromas. These had at first been diagnosed as fibrosarcomas, but review of the sections showed that they were benign. One of these patients had a pleural effusion and the roentgenogram of the chest showed what was thought to be pulmonary metastasis. Postoperative irradiation was given over the chest and the effusion disappeared. Apparently this was a Meigs' syndrome.* The 3 cases with non-neoplastic diseases were all given preoperative irradiation. One proved to be an ectopic pregnancy, one a tubo-ovarian abscess and the third a simple serous cyst of the ovary. Fortunately these patients were near or past the menopause (40, 53 and 77 years respectively) and no ill effects were noted from the irradiation.

Treatment. Ninety-two patients received postoperative and 7 preoperative irradiation. One patient was not operated upon because of heart failure. Of the 7 patients who were treated preoperatively, 3 had ascites and had been classified as inoperable. Following roentgen therapy, the tumor was reduced sufficiently in size so that operation could be performed and at least a portion of the remaining neoplasm removed. In the remaining 4 patients of the preoperative group, the diagnosis was thought to be obvious. Subsequent findings, however, showed that only 1 had carcinoma while the other 3 had non-neoplastic disease.

Irradiation was given with the following factors and techniques: 200 kv. (peak), 5, 8 or 20 ma., 50 cm. focus-skin distance, 0.5 mm. Cu plus 1.0 mm. Al (half-value layer 0.95 mm. Cu), or Thoraeus filter (half-value layer 1.95 mm. Cu). From 1930 through 1935, the pelvis was irradiated through one anterior and one posterior 20 by 20 cm. or 15 by 20 cm. field. In 1936, two lateral 10 by 15 cm. fields were added. Since then, two anterior and two posterior 10 by 15 cm. fields were irradiatied instead of the single large fields and most patients were treated in this manner. A few, however, were still treated through the large single fields. When there was definite evidence that the patients had upper abdominal or hepatic metastases, these regions were also treated, usually through one anterior and one posterior 10 by 15 cm. field. An occasional 8 by 10 cm. perineal field was added.

A dose of 200 roentgens (air) was given to each of two fields daily for six days a week. The total air dose to each field varied from 200 to 5,500 r. Only 5 cases received less than 1,000 r (air) to each field in a single series, while not more than 2,000 r (air) was given to each field in a single series. Twenty-five patients received two or three series of treatments over periods of from six to twelve months. Three patients were given two or three courses over longer intervals of time because of recurrence.

With one exception, all patients were given what was believed to be the optimum amount of irradiation and all tolerated the treatments well. The exception was a woman who by mistake was discharged after the first day's treatment. We were not influenced by the clinical or pathologic findings in outlining the treatment for these patients. If all other conditions were favorable, we considered ourselves limited only by the tolerance of the skin and other normal tissues. The optimum dose usually meant maximum dose, unless the patient's general condition did not warrant it.

Two patients had received radiation treatment prior to admission to the University Hospitals. One woman with recurrent carcinoma had been diagnosed at operation four years before. She had been treated with intracavitary radium at that

* Meigs' syndrome[5] is the occurrence of pleural effusion and ascites in association with fibroma of the ovary. The reasons for fluid formation are not understood. It recurs promptly after tapping. Removal of the ovarian tumor effects a cure.

TABLE V

TUMOR DOSE IN ROENTGENS

Less than 500	500–1000	1000–1500	1500–2000	2000–2500	2500–3000	3000–3500	3500–4000	4000–5000	5000–6000	6000 plus	Total
1	2	3	9	23	17	8	1	6	6	2	78

time and two years later she had had a carcinoma of the cervix for which she had been given three courses of roentgen treatment and additional radium therapy at another hospital. The other patient had been treated with radiEm for recurrent ovarian carcinoma a year before admission, which was five years after the original operation.

The tumor dose delivered to the ovary or its former location was calculated in 78 patients. They had been measured for anteroposterior and lateral dimensions at the time of treatment. In an effort to make the calculations as accurate as possible, measurements were taken with the treatment cone pressed against the patient, thus simulating the actual conditions under which treatment was given. Backscatter and depth dose figures were taken from the tables published by Quimby.[12] For the anterior and posterior fields, the mid anteroposterior diameter was taken as the location of the ovary. When lateral fields were used, a point 5 cm. lateral to the midline was taken as indicating the position of the ovary. When large anterior and posterior fields were irradiated, the calculated tumor dose refers to the dose delivered throughout the pelvis but where lateral fields were used, the tumor dose refers to that delivered to each ovary or its former site. The dose which reached the contralateral ovary through the lateral fields was not included in our calculations.

The distribution of the tumor dose is shown in Table v. Of the 78 patients in whom the tumor dose could be calculated, only 6 received less than 1,500 r and 15 received less than 2,000 r. Forty patients received between 2,000 and 3,000 r, while the remaining 23 were given between 3,000 and 7,000 r. Most of these patients received two or three courses of treatment and the tumor dose is a summation of that given in all courses.

Results. Table VI indicates the length of survival of all patients. It is of interest to note that 48 patients died within three years following irradiation and, of these, 32 died within eighteen months. Only 9 patients died between three and five years after treatment while the remainder lived five years or longer. Thirty-three patients

TABLE VI

SURVIVAL RATE

Group	0–6 mo.	6–12 mo.	1–2 yr.	2–3 yr.	3–4 yr.	4–5 yr.	5–6 yr.	6–7 yr.	7–8 yr.	8–9 yr.	9–10 yr.	10 yr. plus
I		1	3			1	4	2		4		6
II	11	3	5	5	5	1	6	2	2	1	3	4
III	1	1	4		1							1
IV	5	3	3	2		1		1				2
Unclassified	1											
Benign tumors and non-neoplastic disease								3				2
Total	18	8	15	7	6	3	10	8	2	5	3	15

are alive and apparently without disease. Twenty-eight of these had malignant neoplasms, 2 had benign neoplasms and 3 non-neoplastic diseases. Of the cases with malignant neoplasms, 61 (64.21 per cent) died of the disease, while 4 (4.21 per cent) died from other causes without evidence of residual or recurrent neoplasm. One patient (1.05 per cent) died postoperatively and another (1.05 per cent) died of diabetes insipidus one month after completion of treatment. If no cause of death was reported, we have assumed that the patient died of the disease.

Table VII

FIVE YEAR SURVIVAL OF 95 PATIENTS WITH MALIGNANT NEOPLASM

		per cent
Alive without disease	28	29.47
Alive with disease	0	0
Died with disease	9	9.48
Died without disease	1	1.05
Total	38	40

The tabulations of the 95 cases with malignant neoplasm show that 38 (40 per cent) lived longer than five years (Table vii). Nine of these died with neoplasm after five years. One patient lived for fifteen years and died of what was thought to be recurrence; another patient died of massive abdominal recurrence ten years after irradiation (this was proved at autopsy) while a third patient died of an unknown cause eight and a half years after treatment. The remaining 6 patients died within five to seven years following roentgen therapy. One patient (1.05 per cent) died without evidence of neoplasm after nearly eleven years (this was proved at autopsy).

Ascites is usually considered a poor prognostic sign. It is of interest to note that 4 of the 25 cases in our series with ascites lived longer than five years. Two, classified in Group ii, died sixty-six and sixty-seven months, respectively, following irradiation. The other 2 were classified in Group iv. Of these one is alive and the other died without evidence of neoplasm 130 months following irradiation (proved at autopsy as noted above).

Discussion

The clinical diagnosis of malignant ovarian tumor is difficult, especially while the growth is still limited to the ovary. It is generally agreed, therefore, that the best treatment of these tumors is excision followed by irradiation if the tumor proves to be malignant.

At operation all visible neoplastic tissue, even in obviously incurable cases, should be removed. This reduces the amount of toxic material which will have to be absorbed following irradiation. Taylor[15] has found clinical evidence that small masses are handled more efficiently by roentgen therapy than are larger ones.

At the University Hospitals both fallopian tubes, ovaries and uterus are usually removed. Since we do not apply radium in cases of ovarian neoplasm, we believe it is not necessary to leave the uterus or cervix in place as a radium holder.

Postoperative irradiation is indicated in all cases with malignant ovarian tumors, even though the growth appears to have been encapsulated and removed in toto (Group i). It should be started as soon as the patient's condition permits. This is especially important in cases with ascites because rapid reaccumulation of fluid may be prevented. In cases classified in Groups ii, iii, and iv, irradiation is imperative. Although a few cases classified in these groups in the literature have lived five years or longer without recurrence, the chances of this are small. We advise against irradiation if multiple extra-abdominal metastases are present or if the patient is in poor general condition. However, we consider no tumor too extensive for irradiation if it is still confined to the abdomen and pelvis.

The many histopathologic types which differ widely in radiosensitivity and the development of certain complications make it extremely difficult to predict the future course of the disease. The degree of malignancy of the so-called special ovarian neo-

plasms is not definitely known. Disgerminomas most frequently show malignant tendencies while granulosa cell tumors and theca cell tumors show these tendencies less often. In cystadenocarcinoma, which is the most common type of malignant ovarian neoplasm, the capsule is gradually expanded until it ruptures. This results in implantations throughout the peritoneal cavity and the frequent production of ascites. Other malignant neoplasms, however, may also produce implantations through breaks in the capsule. In fact, we consider ascites as a direct indication of peritoneal implants or metastases, which may also not infrequently be produced through lymphatic spread. Bilateral occurrence of ovarian neoplasm is thought to result frequently from implantations or metastases from the primary ovarian neoplasm.

Since the onset of ovarian cancer is usually insidious, diagnosis is frequently not made until the tumor has progressed beyond the primary stage. This was true in at least 74 cases in our series (Groups II, III and IV), and this does not include several cases from which the surgeon had successfully removed all visible secondary neoplasm (classified in Group I).

If the patient's condition does not allow radical or even partial removal of the tumor, preoperative irradiation should be given. Occasionally this is followed by complete disappearance of the secondary masses. The primary tumor may also be considerably reduced in size but it is rarely sterilized. The residual neoplasm should be removed about six to eight weeks following irradiation. If ascites is present, less fluid is formed as the growth begins to regress under irradiation, and therefore paracentesis is needed less frequently. Often this is one of the first indications that the tumor is responding to the treatment (Healy[4]). If possible, however, biopsy or examination of the ascitic sediment should be attempted before irradiation. Of the 7 cases treated preoperatively, a diagnosis of malignant neoplasm had been made by this method in 3 cases; in 1 case no positive diagnosis could be made although the ascitic sediment was examined repeatedly. The 3 remaining cases had non-neoplastic disease. They could have been spared the inconvenience of radiation treatment had they been operated on first.

Irradiation employed by different writers varied little in the physical factors: 165 to 200 kv. (peak), 0.5 to 2.0 mm. Cu, or their equivalents, and a focus-skin distance of 40 to 80 cm. were used most commonly. Usually the pelvis was irradiated through one large or two small anterior and posterior fields. Occasionally upper abdominal fields were added and one group was irradiated through gluteal and ischial fields as well. There was, however, considerable variation in the dosage and method of treatment. Wintz[18] believes that the full dose should be given within two or three days. During the past ten years, however, the majority of authors have treated their patients with fractional doses over periods varying from two to six weeks. The dosage given is usually stated in total roentgens (measured in air), roentgens (measured on the skin), or percentage of skin erythema dose. Jacobs and Stenstrom[6] believe a 50 to 140 per cent skin erythema dose is sufficient for each of several courses of treatment, while other authors[3,4,7,17] report doses (measured in air) of from 1,000 to 2,500 r to each field. Only three observers mention the dose delivered to the tumor site. With the concentrated method, Wintz[18] believes it unsafe to give more than 125 per cent skin erythema dose to the tumor. Healy[4] delivers about 2 threshold erythema doses throughout the abdomen and pelvis, and Montgomery and Farrell[10] consider a minimum of at least 1,600 to 2,000 r to the tumor an "adequate dose." We believe, however, that it is impossible to determine an "adequate" tumor dose in the treatment of ovarian tumors, as it is in nearly all malignant neoplasms. We could find no relationship between the number of roentgens delivered to the pelvis or ovarian site and the rate of survival.

As stated above we have been guided

mainly by the skin tolerance in prescribing the optimum dose. It is of the utmost importance, however, to judge each patient individually in outlining a course of irradiation, and this should be flexible enough to allow for any change in the condition of the patient. Up to 1934, we gave most patients two or three smaller courses of irradiation at from three to five month intervals. From 1934 on, the procedure was changed and most patients received only one intensive course. The use of the Thoraeus filter, the changing of the size of the irradiated fields and the addition of lateral fields allowed us to give larger amounts to the skin and also to reach a higher tumor dose.

As mentioned above, comparison of results reported in the literature is difficult and unsatisfactory since there is no uniform clinical and pathologic classification and the number of cases reported is usually small. Walter and his coworkers,[17] in reviewing the literature on patients treated by surgery alone, found that of 104 cases, which could be classified in our Group I, 48.5 per cent lived five years or longer. Only 6.4 per cent of the 94 cases corresponding to our Group II lived five years. No results were given for the inoperable cases but the rate of survival must be expected to be low. These authors also give their own results which are similar to those from the litera-

Table VIII

Author	Groups									
	I		II		III		IV		Total	
	Cases	Survival per cent	Cases	Survival per cent	Cases	Survival per cent	Cases	Survival per cent	Cases	Survival per cent
Wintz, 1932	4	75	44	25			3	33	51	29.7
Heyman, 1932	46	54	36	22	28	25	24	8.3	134	
Taylor, 1934	25		13		6		40		84	8
Montgomery and Farrell, 1934	4	75	2	100			5	0	11	45.4
Schroeder, 1934	27	60	9	25			20	9	56	32.1
Murphy, 1935									92	20.2
Harris and Payne, 1935	1	100	7	43			6	17	14	35.7
Jacobs and Stenstrom, 1937									31	35.4
May, 1939			16	55			35	23	51	31
Crainz and Schmiemann, 1940	38	60.5	41	19.5			52	9.8	131	27.5
Meigs, 1940									64	12.3
Walters et al., 1941 I	5	20	3	0			7	0		
II	5	20							20	10
I	10	50								Inadequate treatment
II	5	20	11	22.2			5	20	31	29 Adequate treatment
Taylor and Greeley, 1942									26	34.6 Adequate treatment
									30	3.3 Inadequate treatment

ture in Group I, but are 0 per cent for our Groups II and IV. The total survival rate was 6.7 per cent. Wintz[18] reports a survival rate of 10 per cent for his cases treated by surgery alone.

The consensus is that postoperative irradiation results in definite improvement of the five year survival rate. Crainz and Schmiemann[1] state that an increase of this rate from 0 to 20 per cent can be expected from postoperative irradiation of cases classified in our Group II. The total five year results reported by various authors during the past ten years are shown in Table VIII. They vary from 8 to 45.5 per cent, but the majority are between 25 and 35 per cent.

TABLE IX

Group	Cases	3 Year Survival	5 Year Survival
I	21	17 (81%)	16 (76.2%)
II	48	24 (50%)	18 (37.5%)
III	8	2 (25%)	1 (12.5%)
IV	17	4 (23.5%)	3 (17.6%)
Unclassified	1	0	0
Total	95	47 (49.4%)	38 (40%)

Most observers concur in their findings of a definite relationship between the clinical stage and the prognosis. Due to the varied number of cases reported, however, there is considerable discrepancy in the reported five year survival rate. The most consistent results are found in the cases classified in Group I—50 to 75 per cent of these lived five years or longer. These figures are comparable to, although slightly higher than, the results of surgical treatment alone for the same group. The reported results for the other groups, although not so consistent, show a definite improvement over those of surgical removal alone.

The results in our series of malignant neoplasms compare favorably with those cited above (Table IX).

SUMMARY AND CONCLUSIONS

(1) One hundred patients in whom a diagnosis of ovarian tumor was made were given irradiation at the University Hospitals, Iowa City, Iowa, between 1930 and 1938. The neoplasms in 95 cases were malignant.

(2) The total five year survival rate of all cases was 43 per cent, that for the cases of malignant neoplasm was 40 per cent.

(3) Irradiation is indicated in all cases of malignant ovarian neoplasm, but this should be given after the removal of as much neoplastic tissue as possible. Preoperative irradiation is indicated only in advanced inoperable cases. If possible, however, the pathologic diagnosis should be established first.

(4) Our results are in accord with those of most authors as reported in the recent literature. In comparison with surgical treatment alone, there appears to be slight improvement of the five year survival rate in Group I. There is, however, a marked improvement in Groups II, III and IV, which comprise over 75 per cent of all our ovarian neoplasms.

(5) It is our aim to give the maximum tumor dose. To achieve this, the skin tolerance dose was given through multiple fields about the pelvis and abdomen unless it was contraindicated by the patient's condition. We are not influenced by the clinical or pathologic classification.

(6) The majority of our cases received a tumor dose of more than 2,000 roentgens with no untoward consequences.

(7) We have found no relationship between the tumor dose and the rate of survival.

(8) This study again emphasizes the need of early recognition of ovarian cancer, since this will result in a greatly increased rate of survival.

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DISCUSSION

Dr. Charles L. Martin, Dallas, Texas. Drs. Kerr and Einstein have demonstrated the palliative effect of roentgen therapy on carcinoma of the ovary in a very convincing manner. My principal interest in their paper lies in their claim that complete cures have been produced in a few recurrent and inoperable cases. They report a series of 28 patients with proved cancer of the ovary alive and free of disease at the end of five years. The Group I cases were probably cured by surgery, but it is interesting to note that 11 of the salvaged cases fell in Group II, and that in 8 of them the tumor dose was only slightly more than 2,000 roentgens. I find it difficult to believe that peritoneal implants can be cured by this dosage, and hope the essayists will correct me if I have misquoted their figures.

Three of the successfully treated patients in Group II received two long series of roentgen treatments, with an average tumor dose of 4,400 roentgens, and 2 patients with inoperable recurrences did equally well with somewhat larger tumor doses, given in a similar manner. These findings seem to indicate that cancer of the ovary may actually be cured by a tumor dose of approximately 4,400 roentgens, given in two series. Certainly this is a worthwhile observation.

Dr. Einstein (closing). To answer the first question by Dr. Martin: We really cannot claim to have "cured" any of our patients with carcinoma of the ovary. We call it five year "survival." The exception might be one patient with papillary cystadenocarcinoma, classified in Group IV. As I have mentioned before, this patient lived for eleven years and at autopsy no evidence of neoplastic tissue was found.

The second question was about the number of cases with spill of malignant cells into the peritoneal cavity, classified in Group II. There were only 3 cases with spill and without evidence of secondary neoplasm and I have no figures on hand to say whether these were among the five year survivals.

His third question was in regard to the time interval between multiple courses of treatment in the cases living five years or longer. None were treated for recurrent carcinoma, and the time interval between the two and possibly three series of treatments was between three and five months.

A ROENTGENOLOGICAL FILING SYSTEM ADAPTED FROM THE "STANDARD NOMENCLATURE OF DISEASE"

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NUMEROUS classifications, filing and coding systems have been devised to make useful roentgenological teaching material rapidly and easily available. The following article does not propound a new method but summarizes experiences at the Department of Radiology of the Oregon University Medical School in the use of an adaptation of the Standard Nomenclature of Disease[3] for coding and filing valuable roentgenograms over a period of two years.

GENERAL REQUIREMENTS AND CONSIDERATIONS OF A FILING SYSTEM

The purpose of any cross filing or coding system is to place the numerous categories of disease into an orderly system suitable for indexing. This may be accomplished by using an alphabetical arrangement, a system of letters, a system of numerals or a combination of these.

In a medical school it is imperative that a comprehensive, flexible and practical system be used whereby examples from all categories of disease may be quickly and easily segregated and filed for teaching purposes.

In the organization of a cross filing system, the principal aim is simplicity and practicability. Individuals who come in contact with it must be able to grasp it readily and obtain the desired information quickly.

The average practitioner will have nothing to do with a system which appears complicated. For him an alphabetically arranged file would perhaps be better. On the other hand, it is difficult to list diseases in this way. Synonyms are numerous; the arrangement of words labile. The method requires infinite watchfulness in the segregation of the letters.

Simplicity is important where office help will do all clerical work. A uniform terminology of disease in conjunction with a practical manual is essential if the system is to function accurately.

A great difficulty in attaining these attributes lies in the fact that the body is not a simple structure and cannot be divided into simple systems. Its complexity requires considerable study before systems which are consistent, logically subdivided and distinctly separated can be defined. Most classifications are inadequate. The experience of one department is not that of another. Their systems, therefore, are not interchangeable. In spite of comprehensive study and painstaking preparation, consistent and persistent revisions and additions by one or several individuals must be made. It is admitted that the average individual is unable to cover the entire field of disease and anatomy in a systematic and logical way.

In the building of a system, the common diagnoses are quickly listed and the categories just as rapidly filled. Where variety is not so great the roentgenologist centers his interest on the occasional unusual finding. These are placed in their special pigeonhole. Later, other cases belonging to an entirely different group are received and relegated to other divisions. Thus, peculiar and unusual findings begin to expand the file.

To be more specific, "Duodenal Ulcer" is soon filled with numerous clear cut entries. "Osteopetrosis" is referred to the secretary to be put under a new head as a prized example. "Diverticulum of the Stomach" finds its own particular space. Some years later after many more entries have been made, the need for an example of

"Marble Bones" for the next morning's rounds arises. Search is diligently made under this heading. The roentgenologist clearly remembers his having filed an example some time previously. "Osteopetrosis" is searched for, but no such heading can be found. "Unusual Bone Pathology" is next searched and re-searched without success. The following afternoon, after the meeting, the example will be found, accidentally, under "Osteoporosis"! The secretary at the time had been unable to recognize the word and not wishing to embarrass the doctor by asking him to decipher his own writing, had entered it under a heading which looked similar to a word she could recognize.

Walker[5] has succinctly summarized the history of a homemade system in these words: "If you arrange a classification for your department it will start out trim and simple. Then there will be additions. You become accustomed to its logical development. It is apt to wind up a hodge-podge of anatomic, etiologic, and technical terms."

It follows, therefore, that any system adopted requires some little study. But the system which requires the least amount of study and therefore allows for rapid receipt of information will be the most desirable.

In our department a revision of the existing system became imperative. The aforementioned failings were prominent. In looking for "Osteogenic Sarcoma of the Femur" there was as much chance to find the material under "Bone Tumors," "Unusual Tumors," "Sarcoma" or "Tumors of the Lower Extremity." With twenty entries, at least, in each category the amount of time needed to find the specific entity becomes apparent.

Several published classifications and codes[1,2,4] were examined and one chosen which was thought would fit our needs. A month's trial, however, revealed the need of new categories of disease and a reclassification of anatomical sites.

THE STANDARD NOMENCLATURE OF DISEASE

At this time our attention was called to the Standard Nomenclature of Disease[3] now published by the American Medical Association. We have taken this system in its entirety without changing one category and are using it as a radiological cross file. We are also using it to cross file our radiotherapy patients.

To date, the Nomenclature is probably the most comprehensive compilation of diseases ever coded. It is being revised constantly by authorities in their fields. The uniformity of its plan makes it an ideal basis for any medical coding system. It has received considerable recognition among hospitals, being used in many record rooms.

The system upon which it is based is well explained in the introduction to the Nomenclature. Two groups of figures designate the disease condition. The first group of three (or more) digits designates the site of the lesion; the second, separated from the first by a dash, the disease entity or etiological agent.

For example:

360–881 .9 Secondary Squamous Cell Carcinoma of the Lung

i.e. 360– Lung

–881 . Squamous Cell Carcinoma

.9 Secondary

This system has several prominent attributes.

Simplicity. The six or more figures in a row may look quite complex. Their division into topographical and etiological categories, however, is logical. In the final analysis it is simple. Each anatomical site has its three digits, the first identifying the anatomical system. It has been elaborately developed. As an example, 200– belongs to the musculo-skeletal system; 300– to the respiratory system, etc.

The etiological classification is similarly segregated, so that, –100 is designated as, Diseases Due to Lower Plant or Animal Parasites; –400, Diseases Due to Trauma or Physical Agents.

Comprehensibility. The Nomenclature embraces the entire medical field. It, therefore, must include all roentgenological diagnoses of disease. The plan is such that new diseases can always be added without disturbing the existing order.

Flexibility. The system may be as general or elaborate as necessary.

As an example, a "Simple Impacted Fracture of the Lateral Condyle of the Tibia" may be filed under any of the following code numbers:

200–416	Simple Fracture of Bone (Impacted, of Tibial Condyle)
237–416	Simple Fracture of Tibia (Impacted, of Lateral Condyle)
2371–416	Simple Fracture of Upper Extremity of Tibia (Impacted)
23714–416	Simple Fracture of Lateral Condyle of Tibia (Impacted)
23714–416.4	Simple Impacted Fracture of Lateral Condyle of Tibia

The parentheses indicate the detail not specified in the code.

In any event, if the simplest designation is too general it can be expanded, at no time requiring extensive revision of the various entries. In our file we would prefer the second example. If a more elaborate figure is chosen the place of the pathological entity is not greatly removed from its former place. It will be noted that all types of fractures will be found in the same immediate vicinity in the file.

THE SYSTEM IN PRACTICE

Our system is used to classify and arrange only those examples which are useful for teaching and research purposes. Patients of the hospitals and clinics associated with the Medical School receive a unit number which is used on all records, regardless of the number or time of visits. In keeping with this system, all of a patient's roentgenograms are kept in one envelope, identified by the unit number. A copy of all readings of the roentgenograms is typed on the exterior of the envelope. Since they form an integral part of the patient's history, interesting roentgenograms may not be removed and kept in separate teaching or research files.

If, during the reading of roentgenograms a good example is encountered, a note is appended to the original requisition. This note contains the diagnosis and any other pertinent information which may be of value in discussing the roentgenogram at a later date. For example: "Normal Ventriculogram. Excellent filling of lateral and third ventricles. Stereoroentgenograms."

The secretary later adds the code number after reference to the manual, transfers it to an index card and includes the patient's name, unit number, code number, diagnosis and additional notes, the patient's age and date of the roentgenogram (Fig. 1).

Francistine, John 164962 920–000
Normal ventriculogram. Excellent filling of lateral and third ventricles. Stereoroentgenograms. Age 43. 9/6/43.

Fig. 1

The terms used in the manual should be used consistently. The office clerks are then able to make their entries rapidly and easily. Synonyms tend to confuse and result in inaccurate entries.

The cards are placed in order in the file according to the diagnostic code. Suitable divisions are made in the file with different colored tabbed cards for convenience. Further instructions may be obtained from the Nomenclature (1942) pages xii to xv.

In addition, the original requisition is given to the librarian who makes a cross on the patient's envelope and adds the code number. This serves to prevent reduplication of entries.

New diagnoses are coded by the roentgenologist. A record of these is kept for later inclusion within the manual.

The diagnoses at times may be faulty. Additional information, however, is often obtained from ward rounds, staff members and the department of pathology. The unit number aids in locating the chart for still further study. Pertinent information received is added to the index card. In spite of faulty diagnoses the interesting roent-

genogram, at least, is not lost in a sea of routine roentgenograms.

THE MANUAL

The Nomenclature is a sizable volume containing a large number of diagnoses applicable to the entire clinical field. For this reason it is too unwieldy and complex for the radiologist and the office clerks, who are interested only in the less numerous roentgen diagnoses.

DISEASES OF THE GASTROINTESTINAL TRACT, GALLBLADDER AND BILE DUCTS

670– Anus	687—Gallbladder
661– Appendix	
	654—Ileum
682– Bile Ducts	
	653—Jejunum
662– Cecum	
660– Colon	680—Liver
685– Common Bile Duct	
	650—Small Intestine, generally
651– Duodenum	640—Stomach
637– Esophagus	613—Teeth

614–100.2	Abscess, alveolar
661–190.2	Abscess, appendiceal
639–100.2	Abscess, retropharyngeal
613–011	Absence of unerupted teeth
6..–887	Adenocarcinoma of G-I tract
6..–887.0	Adenocarcinoma of G-I tract with metastases
660–887.4	Adenocarcinoma of colon with obstruction
6..–018	Atresia of G-I tract
640–940.9	Atrophic gastritis, chronic
687–923	Calcification in walls of gallbladder
685–615	Calculus in common duct
6..–8y0	Carcinoma of G-I tract, type undetermined
6..–8y0.4	Carcinoma of G-I tract, type undetermined, with obstruction
637–881	Carcinoma of esophagus, squamous cell
631–881	Carcinoma of hypopharynx, squamous cell
640–881.9	Carcinoma, metastatic, squamous cell to stomach
641–580	Cardiospasm, achalasia
641–590	Cardiospasm, reflex
600–x10	Celiac disease
687–615	Cholelithiasis
682–610.6	Dilatation of common duct
6..–630	Displacement of G-I tract
6..–021	Displacement of G-I tract, congenital
6..–630.4	Displacement of G-I tract due to adhesions
6..–642	Diverticula of G-I tract
637–036	Diverticula of esophagus, congenital
637–430	Diverticula of esophagus, traction or pulsion
6..–642.0	Diverticulitis of G-I tract
651–951	Duodenal ulcer
651–951.3	Duodenal ulcer with perforation
651–951.4	Duodenal ulcer with deformity
651–930	Duodenitis
631–555	Dysphagia

FIG. 2

A manual should be constructed, therefore, in which the diagnoses referring to roentgenological conditions are listed. Commonly used anatomical sites should be arranged alphabetically under the anatomical system, the whole forming a handy reference book.

The degree of precision with which a code number is determined would be illustrated in the manual by example. In referring to the foregoing example, a "Simple Impacted Fracture of the Lateral Condyle of the Tibia" would be listed in our file as 237–416 and not 23714–416.4. The latter would burden the file with an excessive number of currently unimportant subdivisions.

In our department the diagnostic codes were determined as the condition arose. After two years all diagnoses were tabulated. Excessively elaborate diagnoses were made more general. Old code numbers were critically examined, revised and unified.

All diagnoses under one system were arranged alphabetically beneath a short pertinent topographical classification (Fig. 2). The latter prevents unnecessary repetitions of diseases as demonstrated by the following example:

6. .–642 Diverticula of G-I Tract

If the correct site happens to be the colon the necessary two figures can be obtained from the above topographical classification, i.e.:

660–642 Diverticula of the Colon

DIFFICULTIES IN CODING

In building a manual, difficulties in coding become apparent. A diagnosis of "Duodenal Ulcer" is simple: its code number quickly determined from the Nomenclature. In the case of "Gastric Ulcer" a complication arises. It must be determined, to some extent, whether the common gastric ulcer or an ulcer the result of neoplasm is meant. If the latter can be demonstrated it should be listed as "Neoplasm of Stomach with Ulcer Formation."

The diagnosis of "Intestinal Obstruction" is not so simple from a coding point of view. It may be listed simply as:

650–610 Obstruction of Small Bowel Due to Unspecified Mechanical Abnormality.

If a more concise designation is required the following may be employed:

650–018 Intestinal Obstruction, Congenital, Due to Atresia
650–100 .4 Intestinal Obstruction Due to Adhesions, Post-infectional
650–611 .4 Intestinal Obstruction Due to Foreign Body
650–639 .4 Intestinal Obstruction Due to Hernia
650–8y0 .4 Intestinal Obstruction Due to Neoplasm, Type Undetermined

In all of the above cases 650– represents the small bowel only.

Whenever an attempt is made to become specific, it should be pursued consistently. Inevitably there arises an intestinal obstruction whose etiology is obscure or indeterminate. To the above list, then, must be added:

650–610 Intestinal Obstruction, Mechanical, Cause Unspecified

A similar condition arises in the classification of the arthritides. To group all arthritides having such widely variant etiologies as infectional, metabolic, neurogenic and unknown, under an all inclusive term would be impractical and illogical.

MANIFESTATIONS VS. DIAGNOSES

Very often the disease itself is not apparent from the roentgenogram. The manifestation, that is, the effect of the disease can be seen. Such appearances as "hydronephrosis," "hydrocephalus" and "mitral configuration of the heart" are not disease entities themselves but evidences of disease. No code numbers are listed under these headings and therefore some difficulty was encountered in transposing them. However, even if the disease entity is not evident, certain generalized facts can be used to form a working code. The cause of a hydronephrosis is not always determinate but the code need only designate an accumulation of secretion in the pelves due to mechanical abnormality, to be sufficiently exact.

Likewise, "hydrocephalus" not due to neoplasia or prenatal conditions was classified:

920–6x8 Accumulation of Secretion in Ventricles Due to Mechanical Abnormality

The code for "mitral heart configuration" was chosen as:

410–196 .0 Rheumatic Heart Disease, Inactive

Although not necessarily correct, it is accurate enough for our purposes. At a future date it may be reclassified. It could be classified, also, under:

454–196 .6 Rheumatic Valvulitis, Inactive, with Deformity of Mitral Valve

or,

417–196 .6 Hypertrophy and Dilatation of Left Auricle Due to Rheumatic Fever

NORMAL STRUCTURES

In a teaching institution a large bulk of the reference file is composed of examples of the normal. The Nomenclature, chiefly interested in disease, has no designated code for the normal. The 1938 edition of the Nomenclature, with which the system was inaugurated, contained the entry:

y00–000 Disease, None. *Change First Digit to Indicate Suspected System, if Any.*

A similar entry is found on page 615 of the 1942 edition. This was used as a basis for the normal. A normal mandible would be designated:

219–000 Normal Mandible, or, a "congenitally non-pathological condition of the mandible."

However, this same figure is used to designate "Diseases Due to Abnormality of Bone Development, Generally and Unspecified" (page 61). Dr. Edwin P. Jordan,

editor of the Nomenclature, suggested, in a personal communication, the use of the etiologic digits –y00, Disease Undiagnosed. The term might be confusing, implying the existence of a disorder which could not be proved from the roentgenograms. Apparently, some discrepancies exist which can be easily worked out.

CONCLUSIONS

We are advocating the use of an adaptation of the Standard Nomenclature of Disease for filing roentgenograms of interest. It would seem logical that a group of roentgenologists, well versed in the requirements of such a system should meet and direct the compilation of a comprehensive manual of roentgenological diagnoses. It would be well if this were published and made available to roentgenologists. Such a manual in conjunction with the Standard Nomenclature of Disease would then be a basis and an aid for the organization of their own indices and files.

SUMMARY

1. A comprehensive, practical, flexible and simple filing system for roentgenograms of unusual interest would fill a definite need. The ordinary attempts are a mixture of etiological, anatomical and technical terms.

2. Difficulties in building a file arise because of the complexity of human diseases and the inadequacy of simple descriptive terms.

3. The system in the Standard Nomenclature of Disease is practical and adaptable to many specialties. It divides the disease entity into an anatomical site and an etiological factor. The same system can be adapted to roentgenological diagnoses.

4. An adaptation of the Nomenclature which has been in use for two years is described.

5. A manual which included the majority of the more common roentgenological diagnoses was compiled from the Nomenclature.

6. Manifestations in contradistinction to diagnoses were coded under more general diagnoses.

7. Since the normal structures have no definite code numbers in the Nomenclature the use of –000 and –y00 following the anatomical sites is suggested.

8. A manual of roentgenological diagnoses adapted from the Nomenclature and compiled by a competent group of radiologists would be of considerable value to roentgenologists of limited practice and of large departments in teaching institutions.

I wish to acknowledge gratefully the helpful suggestions made in the preparation of this manuscript by William Y. Burton, M.D., Assistant Professor of Radiology.

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Vol. 53, No. 4

THE BEHAVIOR OF THIMBLE CHAMBERS WHEN USED FOR THE MEASUREMENT OF VERY SOFT RADIATION

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THE purpose of this paper is to report an investigation of the behavior of a regular Victoreen thimble chamber at wavelengths that, until recently, were longer than the radiation available to the roentgen therapist.

The literature of the last few years includes several papers reporting the physical characteristics of this type of radiation. These studies are reports on radiation produced by contact roentgen therapy equipment. Informative papers covering the subject have been provided by Braestrup and Blatz,[1] Lamerton,[3] and Quimby and Focht.[4] The latter described very thin experimental extrapolation chambers made entirely of lucite.

A review of the literature and a study of the equipment available for contact roentgen therapy indicates that there are in use two types of equipment producing radiation of quite different physical qualities.[2] One of these, the Chaoul type, operating at 60 kv. with no filter, produces radiation having a half-value layer of 3.3 mm. of aluminum, which certainly cannot be called soft radiation. The Philips type equipment operating at 44 kv. with no filter produces radiation having a half-value layer of 0.3 mm. of aluminum, which is appreciably softer than that available from normal types of roentgen therapy tubes.

The chambers supplied with the Victoreen condenser r-meter have not been recommended for use in the measurement of radiation of longer wavelength than that produced by a Universal Coolidge tube operated without filter at 60 kv. Such radiation has a half-value layer of about 0.5 mm. of aluminum, which is approximately 0.4 to 0.5 angström effective wavelength. When used for the measurement of radiation of this quality a regular Victoreen thimble chamber will read low by approximately 5 per cent.

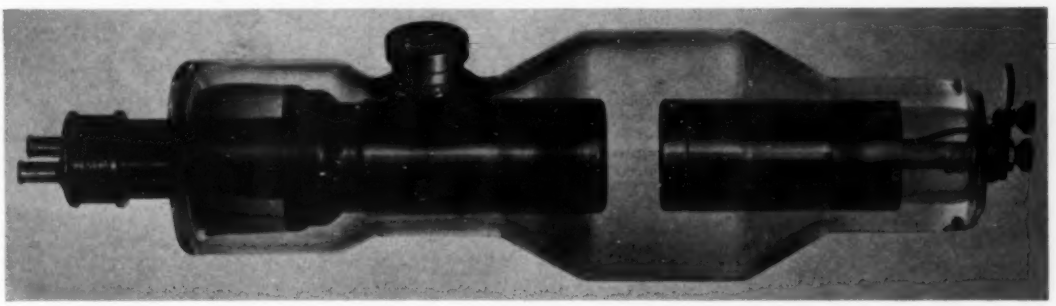

FIG. 1. Hooded anode roentgen tube with beryllium window.

When a conventional (red) Victoreen thimble chamber with a bakelite wall is used for the measurement of the very soft radiation produced by the Philips type contact roentgen therapy unit, both practical and theoretical considerations point to the fact that it will give a reading considerably below the true intensity in roentgens.[8]

The major reason for an investigation of the behavior of thimble chambers at very long wavelengths is the possibility of roentgen therapy tubes having lower inherent filtration making available longer wave-

lengths for treatment. Such tubes have been produced experimentally[5,6] and they may become generally available after the war.

A tube having a beryllium window and characteristics permitting operation in air

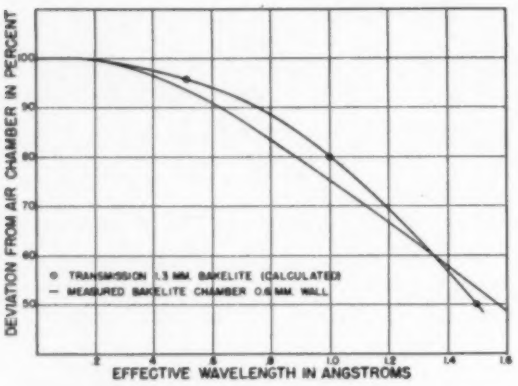

FIG. 2. Wavelength dependence of a regular Victoreen thimble chamber over a wide range of wavelengths.

at voltages from 10 to 160 kv. (peak) was constructed for these experiments. This tube was energized by a Villard type high voltage transformer.

An air chamber designed for very soft radiation was used for all the comparative measurements.

The quality of the radiation in all cases was measured as the effective wavelength. These determinations were made using a material thickness approximately equal to the half-value layer. Copper was used for these measurements up to an effective wavelength of about 0.3 A, aluminum from 0.3 A to approximately 1.0 A, and beryllium for effective wavelengths longer than 1.0 A.

The first data taken were for the purpose of plotting the wavelength dependence of a regular Victoreen thimble chamber at wavelengths up to 1.5 angströms. The results are shown in Figure 2.

From this it will be seen that the Victoreen chamber will be down by about 40 per cent at an effective wavelength of 1.3 angströms and about 50 per cent at an effective wavelength of 1.5 angströms. This deviation from a standard air chamber is largely due to absorption in the bakelite wall of the thimble chamber. It should be remembered that these data deal with very soft radiation. This is readily seen from the plotted data calculated[7] for monochromatic radiation through a flat bakelite sheet only 1.3 mm. thick.

To study the clinical significance of data taken at the very long wavelengths, additional studies were made at an effective wavelength of 1.5 angströms.

From Figure 3 it will be seen that the regular Victoreen thimble chamber is not suited for use in measuring radiation of very long wavelengths. If the radiation therapist uses the intensity indicated by the thimble chamber, the patient will be given a greater dose in roentgens than was indicated by the measurements. The deviation from the true dose increases as the wavelength is increased beyond the range over which the regular Victoreen chamber is supposed to be used. Because of the wide variations in the electrical factors in use and the difficulty in obtaining accurate quality measurements outside the laboratory, it is not possible to set up a table of correction factors that might be useful within clinical limitations.

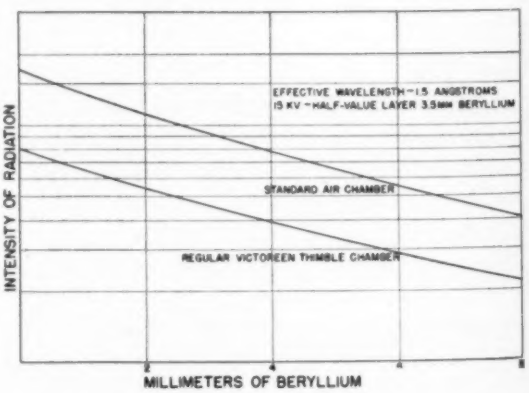

FIG. 3. Absorption curves in beryllium for effective wavelength of 1.5 angströms.

When the regular Victoreen thimble chamber is used for the plotting of transmission data, at very long wavelengths the deviation from the air chamber is not great,

if the beam is fairly well filtered as it emerges from the tube. In Figure 4 the data used in plotting Figure 3 have been plotted, this time in the form of transmission curves. While transmission curves are of value in determining the quality of radiation, they cannot be used in establishing dosage values since the intensity is not stated in absolute values.

An attempt was made to design a thimble chamber that would coincide with the standard air chamber at very long wavelengths. A number of experiments were conducted in the limited time available. Up to the present, the most promising data have

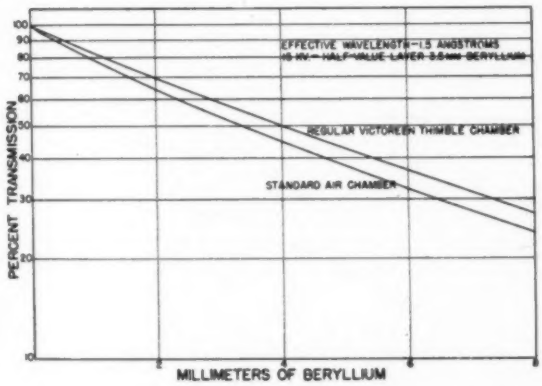

Fig. 4. Transmission curves in beryllium for effective wavelength of 1.5 angströms.

been taken on a standardized thimble chamber with a beryllium wall. The wavelength characteristics for this chamber are shown in Figure 6.

This information on the beryllium chamber is not to be considered as final data on a chamber of this design. A further investigation of this problem is under way and will be the subject of an early report.

The authors are indebted to Mr. Robert E. Rinaker for assistance in many of the necessary calculations.

SUMMARY

The trend toward lower inherent filtrations by the use of beryllium windows in roentgen tubes makes necessary the study of the behavior of thimble chambers for very soft radiations. The standard thimble

Fig. 5. Photograph of conventional Victoreen thimble chamber and experimental Victoreen chamber with beryllium wall.

chamber with red bakelite wall is not suited for measurement of wavelengths much longer than 0.5 angström effective, and at 1.5 angströms it reads low by 50 per cent. An experimental beryllium wall thimble chamber is described with much less wavelength dependence, being only 10 per cent low at 1.5 angströms by comparison with a standard air chamber.

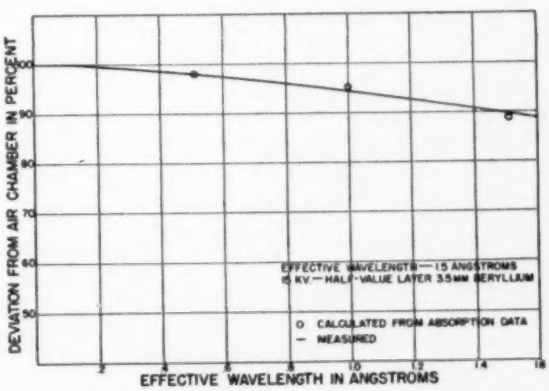
DEVIATION FROM AIR CHAMBER IN PERCENT
EFFECTIVE WAVELENGTH—1.5 ANGSTROMS
15 KV.—HALF-VALUE LAYER 3.5mm BERYLLIUM
O CALCULATED FROM ABSORPTION DATA
— MEASURED
EFFECTIVE WAVELENGTH IN ANGSTROMS

Fig. 6. Wavelength characteristics for an experimental Victoreen standardized thimble chamber with beryllium wall.

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Twenty-eighth Annual Meeting: 1945, canceled.

EDITORIAL

HEMOPTYSIS IN MITRAL STENOSIS

HEMOPTYSIS is a not infrequent complication of rheumatic heart disease. This hemoptysis may accompany acute exertion, upper respiratory infection or it may occur during auricular fibrillation. Investigators on the subject of hemoptysis in rheumatic heart disease all agree that the intrapulmonary blood pressure is elevated. Many explanations have been advanced as a causative factor in the hemoptysis though a satisfactory one or at least one that would explain all the features of hemoptysis has not been found. Some investigators have thought that the hemoptysis is due to pulmonary congestion and hypertension which usually accompany mitral stenosis, causing rupture of the pulmonary blood vessels, or that there is an associated pulmonary arteriosclerosis which weakens the pulmonary vessels. Some think that the bleeding comes from sudden dilatation of the pulmonary capillaries, with diapedesis into the alveoli. However, none of the theories advanced for the cause of the bleeding has been entirely convincing.

Quite recently Ferguson and his collaborators[1] have described a method of injecting the bronchial veins, using a modification of the white lead solution which had been previously recommended by Schlesinger[2] for injecting the coronary vessels. These observers claim to have demonstrated a direct venous connection between the bronchial and pulmonary veins. From their injection methods of study they suggest that in mitral stenosis the veins in the submucosa of the larger bronchi dilate as a result of the collateral flow through them, and that it is the rupture of these dilated veins that leads to hemoptysis.

That communications between the pulmonary veins and the bronchial veins exist has been formerly described and Miller[3] in a series of brilliant studies demonstrated these communications by injection experiments. Ferguson and his collaborators therefore decided to investigate these communications in cases of mitral stenosis in order to see whether submucous varices were present. Since the bronchial veins are difficult to locate and inject, they made their injections into the pulmonary veins with particulate matter too coarse to enter the capillaries. They used necropsy specimens from control cases having no clinical or pathological evidence of mitral stenosis. Six cases were of non-rheumatic heart failure, and eleven cases of mitral stenosis were investigated. All the lungs were from fresh autopsies. The injection mass was introduced into the pulmonary veins at a pressure of 80 mm. Hg. The solution was never seen in the pulmonary capillaries nor did it appear in the bronchial or pulmonary arteries, but in all the cases, including the controls, it was seen to pass freely from the pulmonary to the bronchial veins. In the cases of non-rheumatic heart failure three showed slight dilatation of the veins of the larger bronchi. Pulmonary arteriosclerosis did not affect the flow through the bronchial veins, nor was it affected by age or hypertension without heart failure. Six of the eleven cases of mitral stenosis that were injected showed definitely enlarged bronchial veins, and in four of these the venous dilata-

[1] Ferguson, F. C., Kobilak, R. E., and Deitrick, J. E. Varices of the bronchial veins as a source of hemoptysis in mitral stenosis. *Am. Heart J.*, 1944, *28*, 445–456.

[2] Schlesinger. Quoted by Ferguson, Kobilak and Deitrick.

[3] Miller, W. S. The Lung. Charles C Thomas, Springfield, Illinois, 1937.

[4] Haemoptysis in mitral stenosis. *Lancet*, Jan. 27, 1945, *1*, 122.

tion was pronounced. Four of these eleven cases had a history of hemoptysis and three of these showed pronounced dilatation. Hemoptysis was associated with pulmonary infarction in only one case: one of those in which dilated bronchial veins were demonstrated.

From their injection studies, Ferguson and his colleagues suggest that in mitral stenosis the flow to the left auricle through the pulmonary veins is hindered. As a result of the rise of pressure in these veins the venous anastomosis is reversed and blood passes from the pulmonary veins to the bronchial veins and thence to the right side of the heart. In order to meet this extra strain, the submucosal bronchial veins dilate and become varicose. In their opinion, hemoptysis is then due to rupture or ulceration of these veins, often precipitated by sudden alterations in pressure induced by a severe coughing bout, or by a sudden rise in left auricular pressure such as may occur in auricular fibrillation.

In cases of mitral stenosis in which infarction and acute pulmonary edema are not present, the hemoptysis, so Ferguson and his collaborators believe, is probably due to bleeding from these dilated bronchial veins. While these ingenious injection studies may not explain the whole story, they do shed additional light on the causative factors of hemoptysis in mitral stenosis and their theory fits in fairly well with the current conceptions.

It is through such injection studies as these that many physiological and pathological processes are elucidated. The brilliant work of Batson in his injection studies of the paravertebral veins is a well known example of laboratory experiments explaining observed physiological and pathological processes.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: 1945, canceled.

American College of Radiology
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio. Annual meeting: 1945, canceled.

Arkansas Radiological Society
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1945, to be announced.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Section on Radiology, California Medical Association
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

Radiological Section, Los Angeles County Medical Association
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Chicago Roentgen Society
Secretary, Dr. F. H. Squire, 1754 W. Congress St., Chicago 12, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

Denver Radiological Club
Secretary, Dr. A. Page Jackson, Jr., 1612 Tremont Place, Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida Radiological Society
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meets annually second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

Radiological Society of New Jersey
Secretary, Dr. H. R. Brindle, 501 Grand Ave., Asbury Pk. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

New York Roentgen Society
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N. C. An-

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

nual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport. Next annual meeting, Hotel William Penn, Pittsburgh, May 5–6, 1945.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. L. M. J. Freedman, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary Dr. A.M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Carlton L. Ould, University of California Hospital, San Francisco 22. Meets monthly on the third Thursday at 7:45 P.M., first six months of the year at Lane Hall, Stanford University Hospital, and second six months at Toland Hall, University of California Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. Asa E. Seeds, Baylor Hospital, Dallas, Texas. Next annual meeting, Temple, Texas, Jan. 17, 1945.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly, third Thursday evening.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

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Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. J. W. McKay, 1620 Cedar Ave., Montreal, P. Q.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

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Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Peruana de Radiologia
Secretary, Dr. Victor Giannoni, Apartado, 2306, Lima, Peru. Meetings held monthly except during January, February and March, at the Asociación Médica Peruana "Daniel A. Carrión, Villalta, 218, Lima.

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Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

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Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

To the Editor:

It is with sincere pleasure that we have noticed and welcomed the regular attendances of an increasing number of U.S.A.M.C. roentgenologists at the scientific meetings of our three Radiological Societies—the British Institute of Radiology, the Faculty of Radiologists and the Section of Radiology of the Royal Society of Medicine.

Usually, the three Societies hold their meetings on the third Thursdays and Fridays in each month, in London.

We write as the Presidents of the three Societies to express the appreciation we have all felt of the interest shown and of the part played in discussion by our American radiological colleagues, and to say that their continued attendances will be warmly welcomed whilst they remain in Britain. Many of us wish that we could have offered more private hospitality, but this has been difficult to achieve because of the numerous war-time restrictions.

Our American visitors have undoubtedly helped towards the continued success of our meetings, which are by no means easy to arrange because of the extreme pressure of work on radiologists at the present time, and secondarily because of travelling difficulties.

We remain,
Yours sincerely,

Rohan Williams
(British Institute of Radiology)
Ralston Paterson.
(Faculty of Radiologists)
J. L. Grout.
(Section of Radiology, Royal Society of Medicine)

Full information may be obtained from the Secretaries at:

British Institute of Radiology,
32, Welbeck Street, London, W.1.

Faculty of Radiologists,
45, Lincolns Inn Fields, London, W.C.2.

Section of Radiology,
Royal Society of Medicine,
1, Wimpole Street, London, W.1.

SECOND INTER-AMERICAN CONGRESS OF RADIOLOGY

Preliminary announcement has been made of the Second Inter-American Congress of Radiology to be held in Habana, Cuba, January 19–24, 1946, under the presidency of Dr. Pedro L. Fariñas. The program of the Congress will include four official lectures, scientific sessions, and an extensive scientific exhibit. It is hoped that there will be a large North American delegation present at this Congress and that it may be truly representative of radiology in the Americas. Inquiries for further information should be addressed to the Secretary-General, Dr. R. Hernandez Beguerie, Calle 23 No. 411, Vedado, Habana, Cuba.

CANCER TEACHING DAY

A Cancer Teaching Day presented under the auspices of the County of Erie Medical Society, Buffalo Academy of Medicine, Eighth District Branch of the Medical Society of the State of New York, University of Buffalo School of Medicine, Medical Society of the State of New York, and the New York State Department of Health, Division of Cancer Control, will be held in the Fillmore Room, Hotel Statler, Buffalo, New York, Thursday, April 26, 1945. The meeting will be called to order at 4 P.M. The following two papers will be presented at the afternoon meeting: "Diagnosis and Surgical Treatment of Carcinoma of the Breast," by Donald Guthrie, M.D., Robert Packer Hospital, Sayre, Pennsylvania; "What Can the General Practitioner Do about Lowering Cancer Mortality?" by Lloyd F. Craver, M.D., Memorial Hospital, New York, Dinner will be served at 6.30 P.M. at the Hotel Statler. The evening meeting will begin at 8 P.M. and the following papers will be given: "The Diagnosis and Curability of Intraoral Cancer," by Hayes Martin, M.D., Memorial Hospital, New York; "Carcinoma of the Colon," by John H. Garlock, M.D., Mt. Sinai Hospital, New York.

AMERICAN RADIUM SOCIETY

The annual meeting of the American Radium Society which was planned for June, 1945, has been postponed indefinitely by decision of the Executive Committee. All officers, board representatives and committee members will continue to serve until a meeting of the Society can provide another election.

The following are the new members of the American Radium Society elected by the authority given to the Executive Committee:

H. Milton Berg, M.D., Bismarck, North Dakota
Daniel Catlin, M.D., New York, New York
L. K. Chont, M.D., Winfield, Kansas
George W. Chamberlin, M.D., West Reading, Pennsylvania
Frederick V. Emmert, M.D., St. Louis, Missouri
Gilbert W. Heublein, M.D., Hartford, Connecticut
Leonidas Marinelli, D.O., New York, New York
Alexander Ovalle, M.D., New York, New York
Lewis C. Scheffey, M.D., Philadelphia, Pennsylvania
Dominic J. Verda, M.D., St. Louis, Missouri
Palmer E. Wigby, M.D., Houston, Texas

CLEVELAND RADIOLOGICAL SOCIETY

In commemoration of the semi-centennial anniversary of Roentgen's discovery of the x-ray, exhibits and lectures are being jointly planned by the Cleveland Radiological Society and the Museum of the Cleveland Medical Library. Dr. Otto Glasser, eminent Cleveland physicist and friend of Roentgen, is conducting the arrangements.

The following are the officers of the Cleveland Radiological Society: *President*, Dr. John O. Newton; *Vice-President*, Dr. J. Robert Andrews; *Secretary-Treasurer*, Dr. Don D. Brannan.

DENVER RADIOLOGICAL CLUB

At a recent meeting of the Denver Radiological Club the following officers were elected for the coming year: *President*, Dr. George Unfug, Pueblo; *Vice-President*, Dr. John H. Jamison, Denver; *Treasurer*, Dr. Leonard G. Crosby, Denver; *Secretary*, Dr. A. Page Jackson, Jr., Denver.

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave. Charleston, S. C.

AN IMPROVEMENT IN DESIGN OF THE STEREOSCOPIC PHOTOROENTGEN UNIT

By LIEUTENANT COLONEL ZOLTON T. WIRTSCHAFTER
Medical Corps, Army of the United States

THE necessity for a more rapid and efficient method of loading, unloading, and processing film for the photoroentgenoscope has arisen because of the tremendous number of permanent photoroentgen records required by the Armed Forces. The photoroentgenoscope records two 4 by 5 inch images stereoscopically on one 4 by 10 inch cut sheet of film. Sharpness and detail of the images recorded is equal to that obtained on the usual 14 by 17 inch films. The stereoscopic views clarify doubtful shadows and throw small shadows obscured by the ribs in one view into the rib interspace on the other view. Diagnostic accuracy as checked by many observers has been shown to be at least equivalent, if not superior, to that obtained on the 14 by 17 inch films. In surveys involving large numbers of chest examinations, the ease of operation, processing, and storage of the photoroentgenograms must also be considered. To decrease to a minimum the time consumed by the technical staff in loading and unloading film holders, and in processing large numbers of films manually, a roll film magazine was adapted to the photoroentgenoscope.

In the development of this adaptation, special attention was directed to availability of materials. It must be noted that this instrument was developed for the use of the Air Forces. In the experimental model of this device, a standard aerial camera was modified to replace the cut film holder supplied with the standard unit. The roll film magazine of this camera is detachable and accommodates 56 feet of 5½ inch film at one loading. Due to the lack of sharpness and clarity experienced in using aerial photo films it was necessary to utilize standard single-coated roentgen film developed and recommended for use with the photoroentgenoscope. The camera is equipped with a motor-driven film transport which is actuated by a solenoid. Approximately 75 stereoscopic chest examinations can be recorded on one roll of film. The remote operation of the camera eliminates the need of a technician to reload the film plate holder after each examination.

The entire roll is developed in the Smith aerial roll film developing unit. This unit consists of three telescoping tanks for developer, rinse water and hyposolution. The film is loaded on a reel assembly which is then lowered into the tank of developer. A motor winds the film from one reel to the other, drawing the film through the developer. Upon completion of development, the entire assembly is lifted out of the developer tank and placed in the rinse water. The same procedure is followed for the fixing and the final washing operation. The roll is then dried on the Smith type aerial roll film dryer. Both the developing unit and the drying unit are standard Army Air Force equipment and are available at any base photo unit. Since the technique of developing roll film in this fashion has been developed by the Air Forces, no difficulty is foreseen in this procedure. The total time

necessary for the processing of a roll 56 feet long is one hour and fifteen minutes. This must be compared to the loading, unloading, and processing time for 75 sheets of 4 by 10 inch film. The roll may be stored intact, or it may be cut and each stereo pair filed with the individual's record.

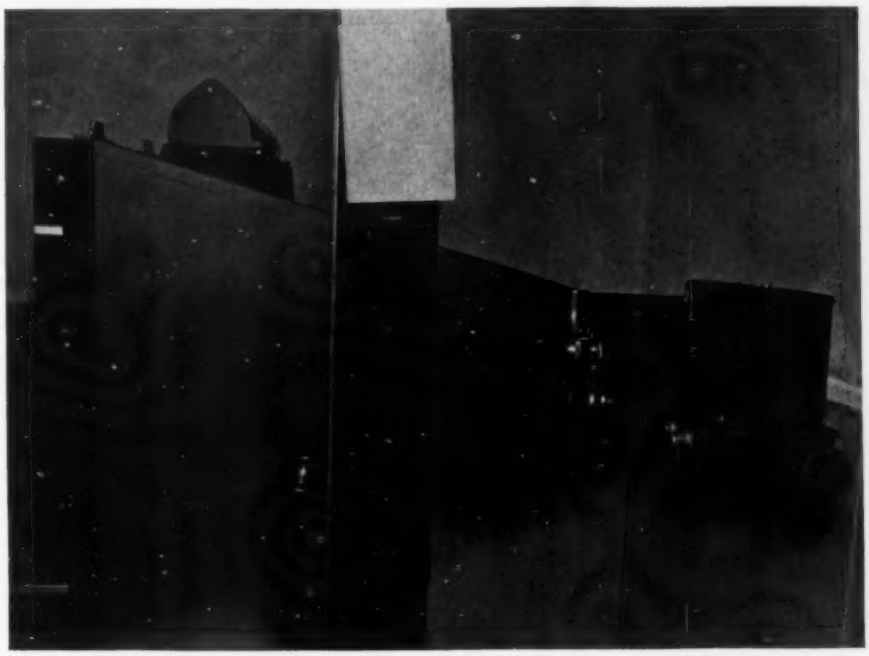

Fig. 1

The advantages of this modification are mainly concerned with the decrease in time of processing, the decrease in personnel, the saving of time in actual operation of the photoroentgen unit, and the elimination of large darkroom area allocated to drying facilities. In addition, it has been found that the photoroentgenograms processed in this manner actually have a smaller percentage of residual hypo than the ones processed in the conventional manner. This assures longer life and a more permanent record.

Although originally intended for use in the Army Air Forces, this equipment could be used by civilian agencies by establishing a central film processing station. It has been suggested that this unit may find application in the field of inspection of small machine parts.

The author wishes to express his appreciation to the Photographic Engineering Branch of Technical Data Laboratory, and to the Photographic Laboratory, Materiel Command, Wright Field, for their excellent cooperation in the development of this apparatus.

A PLASTIC ROENTGEN FILM EXPOSURE HOLDER

By MAURICE M. POMERANZ, M.D.
NEW YORK, NEW YORK

THE Eastman folder for the use of non-screen film is a simple and useful device for its purpose. Its chief disadvantage, however, is that it soils quickly and bends or breaks at the corners, necessitating frequent replacement. It occurred to me that a holder could be constructed of some plastic material which would eliminate these objections.

There are a number of inexpensive plastic materials on the market which can be utilized for this purpose. As an experiment I constructed three folders, one made of bakelite, a second of formica and a third of nitrate sheets. They all served their purpose well. To date, I have not found a sufficient difference in the absorption of these plastics to warrant any modification of technique.

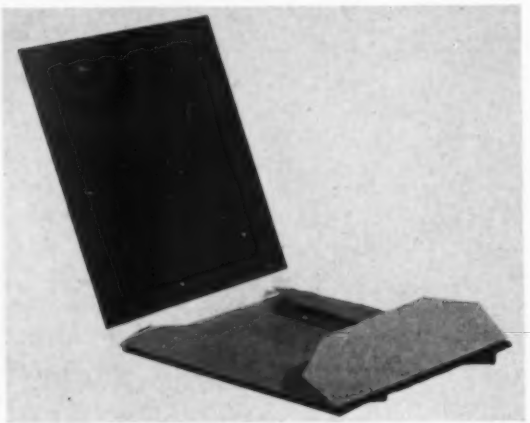

FIG. 1

FIG. 2

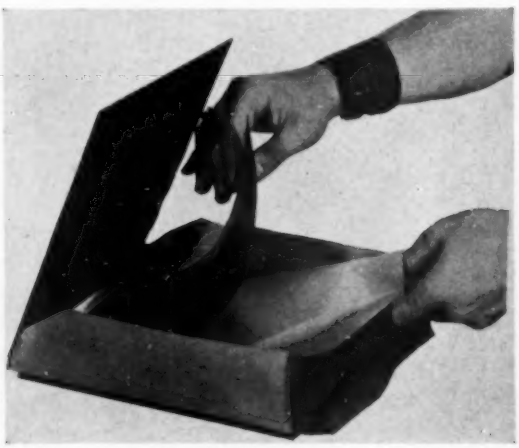

FIG. 3

To construct the folders, two pieces of plastic material about $\frac{1}{16}$ inch in thickness and measuring $8\frac{3}{8}$ by $10\frac{3}{8}$ inches are joined together at one end by adhesive tape which serves as a hinge. On one surface of the plastic sheet, lead foil is cemented or glued. This becomes the back of the holder. To it is next attached the red envelope which is part of the Eastman holder and into which the film is placed when ready for exposure. When attaching the envelope, it should be placed so that the large flap opens upward towards the front cover. Two metal clips or

fasteners are attached to the free end of the holder so that it is effectively sealed when the clips are turned back (Fig. 1, 2 and 3).

A new holder now being made will have two small metal hinges and an inside envelope which will be made of thin black linen.

1160 Park Ave.,
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INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Skeletal System

Berkman, E.: Etiological possibilities of march fractures 407
Cave, E. F.: Calcification in the menisci 407
Milgram, J. E.: Tangential osteochondral fracture of the patella 407
Ghormley, R. K., and Dockerty, M. B.: Cystic myxomatous tumors about the knee 408
Hutchison, R. G.: Osteochondritis dissecans. 408
Brailsford, J. F.: Osteogenesis imperfecta... 408
Phalen, G. S., and Ghormley, R. K.: Osteopathia condensans disseminata associated with coarctation of the aorta 409
Hermon, R.: Two cases of osteochondral hypothyroidism 409
Russo, P. E.: Chondro-osteodystrophy: Morquio's disease 409
Hilliard, C.: Case of chondro-osseous dystrophy with punctate epiphyseal dysplasia.. 410
Herzmark, M. H.: Herniation of cauda equina following laminectomy of the sacrum 410
Oppenheimer, A.: Paravertebral abscesses associated with Strumpell-Marie disease... 410
Perlman, R., and Freiberg, J. A.: Bridging of vertebral bodies in tuberculosis of the spine 411
Halberstaedter, M.: Familial vertebral dystrophy 411
Hyndman, O. R., Steindler, A., and Wolkin, J.: Herniated intervetebral disk 411
Bucy, P. C., and Speigel, I. J.: Unusual complication of intraspinal use of iodized oil.. 413
Samuel, E.: Significance of some developmental abnormalities of the hip joints 413
Mansi, R. L.: Case of insufficiency fractures occurring in neck of femur 414

Blood and Lymph System

Wolcott, W. E.: Evolution of circulation in developing femoral head and neck 414
Harper, R. A. K.: Radiological changes in carotid-cavernous aneurysm 414
Sosman, M. C., Levine, S. A., and Bailey, O.: Syphilitic aneurysm of aortic arch with compression and erosion of esophagus and left main bronchus 415
DeBakey, M., Schroeder, G. F., and Ochsner, A.: Significance of phlebography in phlebothrombosis 415
Williams, D. A.: Arterio-sclerosis 416
Flynn, J. M.: Erythroblastic anaemia 416
Falkenstein, D., and Fowler, W. M.: Acute lymphatic leukemia in childhood 416
Howes, W. E., and Levin, B.: Lymphosarcoma 417

General

Gage, H. C., and Williams, E. R.: Radiological exploration of sinus tracts, fistulae and infected cavities 417
Lichstein, J., and Solis-Cohen, L.: Familial tuberous sclerosis (epiloia) without adenoma sebaceum 418
Kerley, P.: Etiology of erythema nodosum.. 418
Williams, E. R.: Calcinosis 419

ROENTGEN AND RADIUM THERAPY

Fleming, J. A. C.: Carcinoma of the thoracic oesophagus 419
Brown, S., Weiss, H. B., Iglauer, S., and Fine, A.: Carcinoma of trachea 419
Fricke, R. E., and Pastore, P. N.: Radium treatment of granular or hypertrophied lateral pharyngeal tonsillar bands 420
Mandeville, F. B.: Roentgen therapy of orbital-pituitary portals for progressive exophthalmos following subtotal thyroidectomy 420
Leddy, E. T.: Roentgen therapy for bronchiogenic carcinoma 420
Dann, D. S., and Koritschoner, R.: Preoperative roentgen therapy of breast carcinoma 421
Brewer, A. A., and Zink, O. C.: Radiation therapy of acute subdeltoid bursitis 422
Barden, S. P.: Healing of radiation fractures of necks of femora 422

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

SKELETAL SYSTEM

Berkman, E. Etiological possibilities of march fractures. *J. Bone & Joint Surg.*, Jan., 1943, *25*, 206–207.

In this paper, 15 cases of march fractures are discussed. They were proved by history, physical examination and repeated roentgenographic studies. The latter showed fourteen fractures involving the second and third metatarsals in the distal third. Ten were transverse; the fracture lines of the remaining four were never seen, but callus formation was noted approximately two weeks after injury. The fifteenth fracture, clearly seen, transverse in type, and involving the middle third, was in a patient forty-two years old. Thirteen fractures were in new recruits who had been inducted not over two weeks before. All 15 patients had been subjected to routine basic training and long hikes. Symptoms of all 15 patients appeared either during a march or ten to twenty-four hours afterward. Pain, lameness and swelling were the three major symptoms. Long hikes probably cause a severe foot fatigue in new recruits, while resistant Army shoes prevent a proper take-off, and produce excessive strain and stress over the metatarsal heads. Loss of the normal range of motion at the metatarsophalangeal joint, probably due to a tight capsule, predisposes to further loss of proper take-off and a resultant load on the metatarsal shafts. All 15 patients showed some loss of voluntary dorsiflexion.

Treatment consisted of using a cane, wearing Army shoes, and light massage and heat. The average stay in the hospital was five weeks. Later on, instructions were given to increase the range of motion of the toes, especially of plantar and dorsiflexion at the metatarsophalangeal joints. Proper shoe adjustments were instituted to correct the abnormal weight-bearing line. Approximately six weeks following diagnosis of march fracture, all 15 patients were doing full duty.—*R. S. Bromer.*

Cave, Edwin F. Calcification in the menisci. *J. Bone & Joint Surg.*, Jan., 1943, *25*, 53–57.

Cave states the purpose of this paper is to point out the fact that calcified areas may occur in the menisci, and may be mistaken roentgenographically for osseous bodies arising from the synovia or articular cartilage, and lying free in the joint. He reports 2 cases with calcification in the menisci which he operated upon. Reference to the condition in the literature is rare.

The diagnosis of the condition can be made only by roentgenograms. The differential diagnosis is important, because, if the calcified shadow in the roentgenogram is thought to be within the meniscus, a useless search through the remaining portions of the joint can be avoided.

In each of Cave's patients, roentgenograms revealed a calcified shadow in the medial and posterior compartment. In the first case, this was reported as being consistent with a loose body in the knee joint. In the second case, the proper preoperative diagnosis was made, namely, injury to the posterior third of the meniscus with calcification. Both patients were relieved by the operation but Cave points out that symptoms in each were due more to the cartilage injury than to the calcified areas. Each patient presented symptoms and signs of internal derangement of the knee.—*R. S. Bromer.*

Milgram, J. E. Tangential osteochondral fracture of the patella. *J. Bone & Joint Surg.*, April, 1943, *25*, 271–280.

Milgram reports 4 cases in order to describe the syndrome of osteochondral fracture of the patella, to urge its early recognition, and to differentiate it clinically and pathologically from the degenerative type of osteochondritis dissecans of the patella, which also results in the formation of free bodies in the knee joint.

The history is usually that of a child or young adult who twists the knee, usually in the extended position. A loud noise, sudden pain and occasionally a distinct dislocation of the patella laterally ensue. The knee fills with blood. Disability is usually complete. When, in the course

of one to three weeks, the swelling begins to subside, and motion is recommended, a free body is found to exist within the joint. If operation is performed early, it will be found that a previously quite normal patella has experienced a purely traumatic avulsion of the cartilage of (usually) its medial inferior quadrant. If operation is not performed early, recurrent episodes of impingement of the free body will eventually necessitate operation. In this late period of the condition, the free body will be indistinguishable from that of an idiopathic degenerative osteochondritis dissecans.

Milgram suggests that the mechanism is one of tangential direction of the stresses involved. The patella momentarily rubs tightly sideways over the outer condyle with sufficient force to score the cartilage of the patella and femur and leave "glacial grooves" on the patella. Occasionally, probably more often than can be demonstrated, a true dislocation occurs. Then the medial border of the patella catches against the prominent edge of the femoral condyle. As the quadriceps pulls the patella back into line, a chondro-osteal layer is ripped off and left behind and the condylar synovial membrane locally is markedly traumatized.

The treatment recommended is prompt operative removal of the free body. As the walls of the patellar defect were rather perpendicular, Milgram thought it advisable to shave the peripheral edges of the defect down to a moderate obliquity. In his cases, free body formation after operation has not thus far occurred.—*R. S. Bromer.*

Ghormley, Ralph K., and Dockerty, Malcolm B. Cystic myxomatous tumors about the knee. *J. Bone & Joint Surg.*, April, 1943, *25*, 306–318.

The authors report 4 cases of unusual mucinous tumors of the knee joint. Two were probably cysts of the meniscus, one having an unusual amount of bone change, one other was probably an unusually extensive development of a parameniscal cyst, and the fourth was a true myxomatous tumor of the meniscus. They suggest that cysts of the menisci apparently are not true neoplasms, but that they represent the end-results of degeneration. Such cysts may sometimes be distinguished from other cysts in the region of the knee joint by the absence of endothelial lining.

The roentgenographic examination in the second case of their series showed swelling of the soft parts about the lateral side of the knee joint. In the fourth case, extensive hypertrophic changes were shown in the roentgenogram about the medial portion of the tuberosity of the tibia.—*R. S. Bromer.*

Hutchison, Robert G. Osteochondritis dissecans; records of some unusual cases. *Brit. J. Radiol.*, May, 1943, *16*, 147–149.

Osteochondritis dissecans usually occurs at the knee joint and most frequently in the internal condyle of the femur. Seven cases are described and illustrated with roentgenograms in which the site of the process was unusual. In 3 cases the lesion was in the knee joint but at an unusual location, 2 cases in the lateral femoral condyle and in the third in the patella. In 2 of the other cases the lesion was in the ankle, in 1 in the base of the proximal phalanx of the right great toe and in 1 in the head of the third metacarpal bone.—*Audrey G. Morgan.*

Brailsford, James F. Osteogenesis imperfecta. *Brit. J. Radiol.*, May, 1943, *16*, 129–136.

Osteogenesis imperfecta is characterized by imperfect ossification and abnormal fragility of the bones of the fetal skeleton. It is familial and associated with blue sclerotics and often with deafness. These cases can be followed up by roentgenography and the changes that take place through the years noted. Nine cases are described and illustrated with roentgenograms.

In the fetal type, ossification does not proceed beyond calcification of the cartilage; the bone therefore cannot withstand the crushing effect of labor and multiple fractures occur. If the infant survives, the bones show lack of density due to insufficient calcium. The ends of the long bones are expanded and there is some flattening of the curvature of the ribs and bowing of the long bones of the lower limbs. Ossification of the upper limbs takes place at almost a normal rate. As puberty approaches the borders of the metaphyses become irregular; multiple rounded islands of non-ossified tissue may be seen in the metaphyses and ends of the diaphyses. The measurements of the ends of the bones are normal but they appear enlarged because the shafts are so slender; they seem to be solid rods of compact tissue with little or no marrow. In some cases the lower limbs have become so deformed that amputation was necessary. The skull bones are thin and the skull is ballooned.

In cases associated with scurvy subperiosteal hematomas occur. One of the cases described was of this type.—*Audrey G. Morgan.*

Phalen, George S., and Ghormley, Ralph K. Osteopathia condensans disseminata associated with coarctation of the aorta. *J. Bone & Joint Surg.*, July, 1943, *25*, 693–700.

A case of osteopathia condensans disseminata is reported which had some of the characteristics of three types of sclerosing osteopathy—osteopoikilosis, osteopetrosis, and melorheostosis—and in which the patient also had an associated coarctation of the aorta.

Roentgenograms of the skull of the patient were reported negative. Those of the chest showed a notching of the ribs diagnostic of coarctation of the aorta. Of the other bones, the left innominate, left femur, left tibia, left fibula, all the bones of the left foot, the right scapula, right humerus, and right carpal bones were revealed as the sites of disseminated regions of bone sclerosis. There was discrete mottling throughout the bones in the carpus and tarsus and in the region of the epiphyses and metaphyses of the left femur, tibia and fibula, and the right humerus. The coracoid process, the neck and the body of the scapula, and the entire left innominate bone revealed similar spotting together with a more diffuse sclerosing process. The diaphyses of the left femur, tibia and fibula however, showed a remarkable generalized sclerosis with marked thickening of the cortices.

The case thus presented the disseminated spherical and elongated regions of increased density in the bones which characterizes osteopoikilosis. Linear striae or streaks of condensation along the shafts of the bones which have been described as occurring in osteopathia condensans disseminata were also present in the patient. The remarkable thickening of the cortices of the right femur and tibia were, however, more characteristic of melorheostosis or of osteopetrosis. The extreme density of the phalanges was more suggestive of osteopetrosis. To the authors' knowledge no exactly comparable case to this one has been described in the literature.

After discussing the possible causation of these diseases, the authors agree with others that all sclerosing osteopathies are probably due to some defect of genetic arrangement with some faulty differentiation and growth of bone from mesenchyme.—*R. S. Bromer.*

Hermon, R. Two cases of osteochondral hypothyroidism. *Brit. J. Radiol.*, July, 1943, *16*, 208–211.

It has long been known that disturbances in the ossification of the epiphyses occurred in cretins but there may be bone changes that can be demonstrated roentgenologically in lesser degrees of hypothyroidism. Two such cases are described and illustrated with roentgenograms —one in a boy of nine and the other in a girl of four.

Schaefer and Purcell say that so-called juvenile chondro-epiphysitis is not caused by vascular changes or inflammation but by hypothyroidism. They suggest that the confusing terminology of these different diseases should be given up and they should all be called osteochondral hypothyroidism.

It is important to differentiate these cases from Perthes' disease as the latter does not yield to thyroid treatment but prolonged immobilization is necessary in order to prevent deformity. In osteochondral hypothyroidism retardation of bone development is the rule. The appearance of the epiphyses varies from a slight granular lack of homogeneity to a coarse or fine stippling or fragmentation. This fragmentation, however, is only apparent while that of Perthes' disease is a true fragmentation. In addition to the apparent fragmentation in osteochondral hypothyroidism, there is a line of cleavage in the capital femoral epiphyses dividing the epiphysis into inner and outer halves. This is probably due to splitting of the relatively brittle bony epiphysis by pressure. There is a greater tendency to this pressure splitting in osteochondral hypothyroidism than in Perthes' disease, and it may lead to extreme deformity. The epiphysis generally does not show the degree of flattening seen in untreated cases of Perthes' disease.

The importance of routine roentgen examination of the skeleton in suspected cases of endocrine disturbance in young patients is emphasized.—*Audrey G. Morgan.*

Russo, Peter F. Chondro-osteodystrophy: Morquio's disease: case observed during pregnancy. *Radiology*, July, 1943, *41*, 42–47.

A case of Morquio's disease is reported in a colored female dwarf twenty-six years of age. She was in the seventh month of pregnancy and came for treatment for severe dyspnea, which was attributed to the pregnancy. Her body was

36 inches long and she weighed 58 pounds. Both Wassermann and Kahn tests for syphilis were positive. At the end of the eighth month of pregnancy the dyspnea was so alarming that she was delivered by caesarean section. She made an uneventful recovery but the child died ten hours after delivery. The child's skeleton showed no abnormalities but that of the mother during life had shown the typical changes of Morquio's disease. All the bones appeared decalcified. Bone trabeculations were poorly developed and thickened. Various degrees of arrest of bone development had occurred.

This disease was first reported by Morquio in 1929 and up to date about 30 or 40 cases have been recorded. So little is known of the underlying cause of the disease that it is known by various names. Morquio believed the disease to be hereditary and believed that consanguinity and hypocalcemia played an important part in it. Most of the cases so far reported seem to follow the mendelian law. These patients are dwarfs.

The roentgenogram shows shortening and distortion of practically every bone of the skeleton. The epiphyses do not ossify and there is delayed ossification and non-fusion of secondary centers of ossification. All joint spaces are widened and the joint surfaces irregular and eroded. The bodies of the vertebrae are narrowed and flattened. All the bones are decalcified and the changes are symmetrical.

In the early stages of the disease treatment is frequently given for rickets. All forms of treatment tried have proved unsuccessful.—*Audrey G. Morgan.*

Hilliard, Cedric. A case of chondro-osseous dystrophy with punctate epiphyseal dysplasia. *Brit. J. Radiol.*, May, 1943, *16*, 144.

A child born June 22, 1942, was cyanotic at birth, suggesting a congenital heart lesion. The upper arms were short and the fingers in flexion. The legs were flexed and could not be straightened. The birth weight was 5½ pounds and there was no gain during the first month. Roentgen examination about three weeks after birth showed the skull large and globular with the bridge of the nose sunken. The vertebrae were deformed and irregularly developed. The upper arm was short with a large spade-like hand with flexed fingers. The joints of the limbs showed stippling due to alternating areas of increased and decreased density. The areas of decreased density appeared to be composed of osteoid tissue rather than true bone.

A number of other cases of dwarfism with abnormalities of bone structure are reviewed briefly. All these are cases of a fundamental deficiency of cartilage; the most extreme form of this defect is achondroplasia and the mildest, congenital dislocation of the hip which seems to be due to a change in the form of the epiphysis as a result of mucoid degeneration of the cartilage.—*Audrey G. Morgan.*

Herzmark, Maurice H. Herniation of the cauda equina following laminectomy of the sacrum. *J. Bone & Joint Surg.*, Jan., 1943, *25*, 197–201.

A case of herniation of the cauda equina through a defect in the sacrum, made to remove lipoidol, is reported. The author emphasizes the need of caution in opening the spinal canal. Some have advocated hemilaminectomy or other osteoplastic operations to overcome this possible complication. Herzmark concludes that defects in the spinal canal are better protected if bone chips are laid across them before closing the soft tissues.

In the case reported, the cord was found protruding from the canal in a loop-shaped segment, caught by a band of fibrous tissue above and compressed against the sharp thin ledge of bone which formed the lower boundary of the defect made in the sacral canal at the time of the removal of the lipoidol. The gap in the neural canal was covered by taking thin, flat pieces of bone from the sacrum, and placing them across the canal like tiles, until the cauda equina and its canal was completely roofed. The patient made a very satisfactory recovery.—*R. S. Bromer.*

Oppenheimer, Albert. Paravertebral abscesses associated with Strumpell-Marie disease. *J. Bone & Joint Surg.*, Jan., 1943, *25*, 90–96.

Oppenheimer could find no references in the literature to Strümpell-Marie disease associated with paravertebral abscesses and he found it difficult to understand why these two distinct conditions were associated. He had 4 cases and some findings were common to all of them. In each the disease was chronic and began with pain and stiffness in some part of the back or neck. In the course of several years, the pain tended to subside and the stiffness in-

creased but the symptoms remained confined to the region originally affected. Abscesses in the soft tissues of the back or neck were present at the level, but not below, the involved vertebrae. Roentgenograms showed an ankylosing arthritis of the apophyseal joints at the level of the soft tissue abscess, with ossification of the vertebral ligaments, but without destruction of bone which might account for the abscesses.

In the 4 cases, roentgenographic examinations failed to reveal signs of bone destruction and repair. In speculating on the association of these two conditions, Oppenheimer could think of only two pathological processes which could account for the lesions observed in his patients. He thought it possible that the lesions were caused by a hitherto unknown type of necrosing bone infection. Since no evidence of necrosis could be found, he considered the vertebral origin of the abscesses as doubtful. Since this was the case, he thought the possibility of the abscesses being the cause rather than the result of the apophyseal arthritis should be considered. He believes the possibility cannot be entirely discarded that the apophyseal arthritis was the result of the persistent hyperemia which accompanied the chronic purulent infection of the paravertebral soft tissues. As in other cases of rheumatoid spondylarthritis, the ossification of vertebral ligaments which was present in his cases was a secondary phenomenon.—*R. S. Bromer.*

Perlman, Robert, and Freiberg, Joseph A. The bridging of the vertebral bodies in tuberculosis of the spine. *J. Bone & Joint Surg.*, April, 1943, *25*, 340–350.

The findings in the active stage of tuberculosis of the spine are well known, namely, bone destruction, osteoporosis, and abscess formation. Healing takes place by fibrosis, organization of abscesses, resorption of sequestra, osteogenesis, and at times bony fusion between the involved vertebrae. This healing is seen in the roentgenogram as sclerosis of bone, sharper delineation of the bones, and decrease in the size of the abscesses. The phenomenon of bridging of the vertebral bodies at the site of the tuberculous bone involvement is not widely known. This bridging in tuberculous spondylitis may be overlooked and has come to be associated rather with pyogenic infection of the vertebrae.

Five cases are reported, 4 showing osseous bridging of the vertebral bodies and the fifth, abortive bony bridging which could not be considered to be actual spontaneous fusion. They regard the process as a reaction to irritation with periosteal new-bone formation or ossification in ligamentous tissue and not as evidence of a healing process. In certain instances such bony bridging accomplishes fusion of the vertebrae involved by tuberculosis as effectively as posterior surgical fusion.—*R. S. Bromer.*

Halberstaedter, Max. Familial vertebral dystrophy. Case reports. *Brit. J. Radiol.*, April, 1943, *16*, 121–124.

Cases of vertebral dystrophy are described in two brothers aged thirty-nine and thirty-five. The parents and a brother and sister are normal. The first patient came for treatment on account of pain in his left hip which was shown to be due to osteoarthritic changes. Examination of the other members of the family followed. The vertebrae are flattened and elongated in the anteroposterior direction. The upper and lower edges of the vertebrae are ill-defined and uneven and the intervertebral spaces are a little decreased. Some of the nuclei pulposi show calcareous degeneration. There is notching just behind the anterior edge in the lateral view. The flattening and elongation of the vertebral bodies is less marked in the lumbar than in the dorsal spine. In both cases there is straightening out of the dorsilumbar curve. In the younger brother the second phalanges are shortened. The pelves are smaller than normal in both cases.

The familial incidence and widespread changes in the spine, together with the changes in the pelvis and hands, indicate that these are cases of familial osteodystrophy. The type of dwarfism, due to shortening of the spine, is quite different from that seen in achondroplasia. Brailsford has described four types of chondro-osteo-dystrophy, and these cases, from the type and pattern of the lesion, the pelvic changes, the slight peripheral changes and the familial incidence, seem to belong in his class C. —*Audrey G. Morgan.*

Hyndman, Olan R., Steindler, Arthur, and Wolkin, Julius. Herniated interveterbral disk; a study of the iodized oil column. *J. Am. M. Ass.*, Feb. 6, 1943, *121*, 390–401.

Herniated intervertebral disk, or herniation of the nucleus pulposus is clearly a causal factor

for low back pain associated with radiating pain in the sciatic distribution. Herniated disk provokes a syndrome of spinal nerve root compression, and the radiating pain is referred pain associated with the signs and symptoms that attend direct root involvement. A syndrome due to myofascial trauma is associated with or provokes a strictly reflex sciatic radiation and hence can be differentiated from referred sciatic radiation. In this paper the authors report on 63 laminectomies with reference chiefly to a method of differential diagnosis and to the interpretation of the iodized oil column. Herniated disks were found in 50 cases. The disk was below the fourth or fifth lumbar vertebra in 46 cases (92 per cent). It was below the seventh cervical in 1 case, below the ninth dorsal in 1 case and below the third lumbar in 2 cases.

All 46 patients with herniated disks at the fourth or fifth lumbar described radiating pain down the posterior aspect of the thigh and at least to and involving the leg—usually the lateral aspect or calf of the leg. Thirteen of these patients said there was no pain in the foot. Thirty-three described pain radiating to the foot—usually the ankle, dorsum or ball of the foot. Coughing and sneezing exaggerated the pain in the back and down the leg in 39 cases.

Signs included spasm of the erector spinae muscles with a list favoring the pathologic side, pain and limitation on bending forward as in touching the floor, reproduction of the radiating pain by forcefully pressing in the paravertebral space between the iliac crest and the lumbar spine with the thumb and on the pathologic side and tenderness along the course of the sciatic nerve and of the calf muscles. Kernig's sign or the straight leg raising test was positive in all cases.

The iodized oil column is characterized at least by pairs of axillary pouches which mark the points at which nerves leave the dural canal. The axillary pouch is the landmark for herniated disk. In all of the cases in this series if the disk caused a notch in the lipiodol column, the notch included the axillary pouch. If a filling defect occurred between two pairs of axillary pouches and did not include either, it was not due to a herniated disk. In analyzing the iodized oil films the defects are classed according to the following types: 1. Complete, in which a filling defect extends completely across the iodized oil column. 2. Definite unilateral defect, in which a definite notch is cut out of the iodized oil column on one side. 3. Definite bilateral defect in which a definite notch is cut out of the iodized oil column on both sides at the same segment. 4. Mild defect, in which there is a mild filling defect at an axillary pouch. 5. Root sign, in which there is no filling defect in the iodized oil column proper but there is some displacement of the root from its normal course or a blunt cessation of the passage of iodized oil down the root sheath.

The phenomenon of referred sciatic pain is most strikingly represented in the clinical syndrome of the herniated intervertebral disk. Sciatic pain due to pressure of the disk on the sensory spinal root is of mechanistic origin. There is, however, another possible source of the pain phenomenon, namely that it is of reflex origin. By this is meant that the pain impulse travels from a peripheral point, in this case the strained soft structures of the back, centripetally to the spinal cord, and makes then synaptic contact with other sensory units at different levels and with different anatomic distribution, with the result that pain sensation is recorded in the territory of the second unit.

The reflex character of such a sciatic pain can be proved only if the reflex arc can be interrupted at the point of the primary local irritation, in this case the soft tissue lesion in the back, and if such an interruption immediately and absolutely abolishes the radiation along the sciatic nerve. In the case of the low back pain this is facilitated by the fact that many patients show so-called trigger points; that is, small, strictly localized and distinctive areas of pain on pressure. These are most frequently the lumbosacral junction, the posterior superior iliac spine, the gluteal insertion at the outer posterior rim of the ilium and the posterior border of the tensor fasciae. For the past five years the authors have been able to prove that some sciatic radiations associated with low back pain are of this purely reflex character. This proof is furnished by producing or intensifying sciatic radiation by simple stimulation of the circumscribed painful area in the back; furthermore, by the fact that infiltration with 5 to 10 cc. of a 1 per cent solution of procaine hydrochloride into this area at once abolishes the sciatic radiation.

In a small percentage of patients who present definite symptoms and signs of root compression, including an absent achilles tendon reflex, no herniated disk, concealed or otherwise, is found. In this type of patient complete removal

of the fourth and fifth lumbar laminal arches and their accompanying ligamenta flava and partial unroofing of the intervertebral canals of the fifth lumbar and first sacral roots are advisable. The results in this type of patient following such a decompression have been very encouraging.—*S. G. Henderson.*

Bucy, Paul C., and Speigel, Irving J. An unusual complication of the intraspinal use of iodized oil. *J. Am. M. Ass.*, June 5, 1943, *122*, 367–369.

Although the intraspinal injection of iodized oil is ordinarily an innocuous procedure, the existence of a lesion which retains the iodized oil in contact with the spinal cord may ultimately result in undesirable changes in the spinal cord. In support of this the authors cite the case of a man aged thirty-six who had had low back pain beginning in 1935. It gradually became more severe and after two years spondylolisthesis was discovered. In April, 1937, a spinal fusion was performed at the fourth and fifth lumbar and the first sacral vertebrae. After this operation the patient felt quite well. However, by the beginning of 1938 he began to have severe pain in the left hip radiating down the left thigh posteriorly. In March, 1938, no neurologic abnormalities were found on examination. Iodized oil was injected intraspinally by the lumbar route and the patient examined on the tilting fluoroscopic table. At this examination no obstruction or filling defect of the spinal canal was seen, but two large globules of oil became caught and remained at the level of the eighth thoracic vertebra. Nothing further was done. There was gradual improvement in the patient's symptoms, and by the end of 1938 he was almost entirely well and able to resume work.

About six months before admission to the Illinois Neuropsychiatric Institute on February 23, 1942, the patient began to notice that both his lower extremities were becoming weak and stiff. Weakness and stiffness progressed to such an extent that it became difficult for him to get about without using the walls and furniture for support. He also noticed that his lower extremities were becoming "numb." About four weeks before admission, the patient noticed a burning pain along the medial aspect of the right foot which came and went for varying periods of time. On lumbar puncture in February, 1942, an almost complete spinal block was found. At operation on March 10 two collections of encysted iodized oil in the subarachnoid space and a very much thickened arachnoid membrane were found and removed. Within a few weeks after the operation the patient had made a nearly complete recovery.

It is concluded that this patient suffered from a localized adhesive arachnoiditis at the level of the eighth thoracic vertebra prior to the injection of the iodized oil. This arachnoiditis caught and held some of the iodized oil, which in turn stimulated fibroblastic proliferation in the leptomeninx, thus increasing the arachnoiditis and resulting in dysfunction of the spinal cord.—*S. G. Henderson.*

Samuel, Eric. The significance of some developmental abnormalities of the hip joints. *Brit. J. Radiol.*, Aug., 1943, *16*, 250–254.

The roentgen appearance of congenital dislocation of the hip is well known but there are some lesser congenital abnormalities about which little is known. These may and very often do lead to osteoarthritis in later life. At first they cause little inconvenience to the civilian but under the greater stress of military life they may cause considerable pain and difficulty in walking.

The technique of examination for these abnormalities is described. With the medial sides of the balls of the two great toes touching each other and the heels 2 inches apart, the degree of anteversion and torsion of the shaft of the femur is shown approximately by the position of the lesser trochanter. The direction of the acetabulum in relation to the coronal plane of the pelvis can also be estimated.

A separate center of ossification often appears at the upper border of the acetabulum and forms a separate bone known as the os acetabuli. This causes no symptoms. It was found in many cases of the author's 107 cases examined for hip-joint symptoms.

Varying degrees of failure of development of the fossa of the acetabulum occur in congenital dislocation of the hip joint. Minor degrees of this failure of development cause congenital subluxation which is not so generally recognized. The roentgen signs are changes in the joint space, the lower part of which is widened while the upper part is narrowed from upward displacement of the shaft, and shallow and poor development of the acetabulum. There is never a very marked degree of upward displacement of the head. There is an apparent coxa valga due to eversion of the leg to compensate for the upward displacement. Theoretically these pa-

tients should be liable to traumatic dislocation but no cases of traumatic dislocation were seen in this series. Another type of congenital dislocation is seen when there is a disproportion between the head of the femur and the acetabular fossa.

Another abnormality frequently seen is increased depth of the acetabulum. Certain markings seen on the roentgenogram around the hip joint, first described by Köhler, are of great importance in estimating the depth of the acetabulum. Sometimes the floor of the acetabulum approaches the internal wall of the pelvis progressively until a true protrusion occurs. According to Gilmour, protrusion of the acetabulum is due to abnormally early fusion of the Y-shaped epiphyseal cartilage on the floor of the acetabulum as a result of which abnormally thin bone is formed in the floor of the acetabulum which weakens under weight bearing and bulges inward. Some cases of protrusion of the acetabulum occur, however, after infectious conditions of the hip joints. A lesser degree of inward bulging causes a deep acetabulum which may contain two-thirds or the whole of the head. The joint space may be abnormally narrow throughout the hip joint. These deep acetabula frequently cause osteoarthritis.—*Audrey G. Morgan.*

Mansi, Ronald L. A case of insufficiency fracture occurring in the neck of the femur. *Brit. J. Radiol.*, April, 1943, *16*, 119–120.

A case of "insufficiency" or fatigue fracture is described in a young soldier nineteen years of age. He came for treatment for slight pain in the right hip when walking. There was no history of trauma. Roentgen examination showed a fine fissure extending about a quarter of an inch into the neck of the femur. The patient was not seen again until about six weeks later when the fracture had extended completely across the neck and was practically healed in perfect position. At this time the patient was free of pain. He had not gone to bed in the interval but had been doing light office work. There was no roentgen evidence of generalized disease of the bone.

It seems surprising that there should be no displacement in this type of fracture. It is possibly because the fracture takes place so slowly that some union takes place in the first part of it before it is complete. There will probably be increasing numbers of this type of fracture in the services and roentgen follow-up of cases with a history of persistent pain is recommended.—*Audrey G. Morgan.*

BLOOD AND LYMPH SYSTEM

Wolcott, W. Eugene. The evolution of the circulation in the developing femoral head and neck; anatomic study. *Surg., Gynec. & Obst.*, July, 1943, *77*, 61–68.

The injection method would seem to be a practical way of studying the vascular pattern in the head and neck of the femur at different age levels. By this method, the author was able to visualize the development of the blood supply which nourishes the ossifying center in the head of the femur during the entire growth period. It also was possible by this method to locate the course of the vessels within the femoral neck and head which nourish the growth area from the beginning through to its completion. This knowledge should prove to be helpful in the approach to a better understanding and treatment of certain hip conditions met in children especially Legg-Perthes' disease and the so-called slipped epiphysis. The author concludes:

1. The ossifying center in the developing head of the femur in infants and children receives its blood supply from the visceral capsule vessels which arise from the median circumflex artery.

2. The ligamentum teres vessels do not enter the head of the femur in children nor do they contribute to the nourishment of the growing femoral head, except for very small vessels which accompany the fibrous tissue at the implantation of the ligamentum teres into the fovea area.

3. The anastomosis between the ligamentum teres vessels, the capsular arteries and the nutrient artery of the shaft does not take place until the ossification of the head of the femur is practically complete, at which time the vessels of the three systems unite by penetrating the thinned out cartilage area at the fovea, thus establishing the anastomosis.

4. The ligamentum teres circulation is a closed circulation so far as the femoral head is concerned until such an anastomosis takes place.—*Mary Frances Vastine.*

Harper, R. A. Kemp. Radiological changes in carotid-cavernous aneurysm. *Brit. J. Radiol.*, May, 1943, *16*, 150–152.

Two cases of carotid-cavernous aneurysm are described and roentgenograms given showing

the bone changes that accompanied them. Thirty-eight spontaneous cases and 80 traumatic cases have been reported in the literature. In 2 of the spontaneous cases similar bone changes occurred; in 1 traumatic case there were changes in the anterior fossa of the skull which may have been due to some other cause.

The changes consist of erosion of the sella with thinning of the lateral wall, and thinning and erosion of the clinoid processes and the sphenoidal ridge with slight enlargement of the sphenoid fissure and optic foramen. In the 2 cases described here such changes had persisted for twenty years and one year respectively after operation for the aneurysm. The degree of bone change depends on the severity of the process, that is the size of the tear in the internal carotid artery inside the cavernous sinus, the time before operation is performed and the degree of success of the operation.—*Audrey G. Morgan.*

Sosman, M. C., Levine, S. A., and Bailey, Orville. [Syphilitic aneurysm of aortic arch with compression and erosion of esophagus and left main bronchus.] *Radiology*, July, 1943, *41*, 64–69.

At this clinico-pathological conference the case of a fifty-two year old unmarried Irish domestic was considered. She came with a clinical picture suggesting aneurysm of the aorta—pain between the scapulae not related to effort, paralysis of the left vocal cord, cough and finally evidence of obstruction of the superior vena cava. Roentgen examination, on the contrary, seemed to show bronchogenic carcinoma. The roentgenograms are reproduced. The blood tests for syphilis were negative. In the belief that it was a tumor and the hope that it might be radiosensitive, roentgen treatment was given. This was followed by dyspnea and deep cyanosis. She became unconscious and had several attacks of this kind during the remaining week of her life. Independent of these attacks, she had two episodes of auricular fibrillation each lasting about four hours. Anemia increased rapidly, respiration gradually failed and the patient died in coma. Autopsy showed syphilitic aneurysm of the arch of the aorta with compression and erosion of the esophagus and left main bronchus. The evidence was pretty evenly matched in this case for aneurysm and tumor and the points in differential diagnosis are considered. The purely clinical diagnosis of aneurysm was obscured in this case by the laboratory tests and roentgen examination which usually prove so helpful.—*Audrey G. Morgan.*

DeBakey, Michael, Schroeder, George F., and Ochsner, Alton. Significance of phlebography in phlebothrombosis. *J. Am. M. Ass.*, Nov. 20, 1943, *123*, 738–744.

Until recently, combative measures in pulmonary embolism have been singularly ineffective. Solution of the problem consists essentially in prophylaxis. The direction of attack must be focused on the prevention of fragments of an already existing thrombus from reaching the pulmonary vascular channels. Obviously the logical means of doing this is by blocking or ligating the venous channel central to the site of the thrombus. The practical application of proximal venous ligation in the prevention of pulmonary embolism has been difficult due to the frequent absence of precise criteria in determining or in predicting the possible occurrence of embolism. For this reason, some have advocated routine division of the femoral vein in all patients who have or are suspected of having thrombophlebitis of the deep veins of the lower leg. In thrombophlebitis the clotting is believed to be the result of injury to the vascular endothelium from mechanical trauma, bacterial invasion or chemical injury, whereas in phlebothrombosis it may be due to venous stasis and to alterations in the cellular and fluid constituents of the blood that increase the clotting tendency. In thrombophlebitis the clot is usually firmly adherent to the vein wall and is therefore less likely to become detached and to result in embolism. In phlebothrombosis, on the other hand, the thrombus is loosely attached to the vessel and is more likely to cause embolism. The development of phlebothrombosis is apt to be insidious and symptoms may be obscure. The patient does not appear so sick as in thrombophlebitis, but restlessness and anxiety are frequently present. Hence, a precise method of diagnosis of intravascular thrombosis is desirable. Herein lies the importance of phlebography.

Phlebography is simple technically and is a relatively safe procedure. The patient is placed on the roentgenographic table on his back with a 7 by 17 inch film under his leg and lower thigh. A tourniquet is applied to the thigh just below the fossa ovalis and just tight enough to occlude the superficial circulation. The leg and thigh are internally rotated in order to separate the shadows of the tibia and fibula and secure unobstructed visualization of the veins. Twenty cubic centimeters of a 35 per cent diodrast solution is injected into any vein on the dorsum of

the foot or ankle at the rate of 1 cc. per second. Twenty seconds after the injection is completed the film is exposed. By employing a tube-film distance of 6 feet in which parallel rays will reach the film, it is possible with two films placed in tandem to visualize the venous system of the entire lower extremity. Twenty-five cubic centimeters of diodrast solution is injected and the exposure is made approximately thirty seconds after completion of the injection.

Normally both the deep and the superficial veins of the leg and thigh fill with the contrast substance and are clearly delineated on the roentgenogram. Incomplete or irregular filling or absence of filling is an indication of thrombosis. If the deep veins are thrombosed, the superficial veins fill and are dilated. If a defect in the venous system is visualized, indicating the presence of a thrombus, steps should be taken immediately to prevent its detachment, either by ligating the vein above the site of the thrombus or by removing the thrombus.

Eleven case histories are given together with illustrative roentgenograms.—*S. G. Henderson.*

Williams, D. A. Arterio-sclerosis; a case report. *Brit. J. Radiol.*, May, 1943, *16*, 142–143.

A woman, aged fifty-two, had had progressive debility for a year and had become very forgetful. Her general condition was poor. On the ring finger of the right hand were two small ulcers caused by the prick of a thorn. There was also a superficial ulcer above the left external malleolus. There was some cyanosis of the toes. There was marked generalized arteriosclerosis and the blood pressure was 170/100 mm. Hg. Wassermann and Kahn tests were negative. The urine showed a few granular casts and some leukocytes and red blood cells. She became drowsy and died in uremia.

Autopsy showed the skull softer than normal with an appearance suggesting decalcifying osteitis. The kidneys were very much atrophied and the capsules adherent. There was intense atrophy of the parenchyma with fibrosis. Most of the tubules that remained were dilated and filled with hyaline casts giving an appearance resembling that of thyroid tissue. The kidneys were shrunken and the vessels thick walled. The aorta and its terminal branches were atheromatous. The coronary arteries were markedly arteriosclerotic. There was extensive calcification of both intima and media. The lungs showed emphysema, edema and bronchopneumonia.—*Audrey C. Morgan*

Flynn, James M. Erythroblastic anaemia with a review of the literature. *Brit. J. Radiol.*, June, 1943, *16*, 157–165.

A case of erythroblastic anemia in a boy of Italian parentage eight years of age is described and illustrated with roentgenograms. He showed definite pallor with a slight icteric tint at eight months of age. As the years passed he developed mongoloid facies, his abdomen became more and more prominent and edema began at the ankles and progressed upward to the upper limbs. He was given seven transfusions in the course of several years. He is now acutely ill with protruding abdomen, pallor, cyanosis of the lips, generalized edema and dyspnea.

This disease occurs usually along the Mediterranean or in children whose parents came from there. It is characterized by enlarged spleen and liver, pallor, icterus, and in the blood picture anisocytosis and poikilocytosis and a peculiar achromia. There is hyperplasia and thinning of the cortex of the bones, the changes in the skull bones causing the mongoloid facies. Target cells are a characteristic finding. These are so called because of their resemblance to bull's eyes or targets. Their significance in the blood picture is not known. Neither is the cause of the disease known though there is some evidence that it is due to damage of the blood-forming system by congenital malaria. It generally begins in early infancy and the children die before adolescence, though a number of cases have been reported in adults. No treatment has been found effective, though liver, cod-liver oil and various medications have failed. Some rather good results and some failures have been reported from roentgen treatment though it has not been tried extensively. —*Audrey G. Morgan.*

Falkenstein, Dorothy, and Fowler, Willis M. Acute lymphatic leukemia in childhood. *Am. J. Dis. Child.*, March, 1943, *65*, 445–454.

The authors reviewed the cases of 61 consecutive patients with acute lymphatic leukemia admitted to the pediatric service. All patients were under sixteen years of age. They found that the aleukemic and leukemic forms of acute lymphatic leukemia in childhood occur with about equal frequency. They present no essential differences in their clinical picture except that the leukemic form tends to pursue a somewhat more acute course and to produce more pronounced lymphoid hyperplasia. The aleu-

kemic form is more difficult to diagnose and is more easily mistaken for aplastic anemia, arthritis or an acute infectious process.

High voltage roentgen therapy was tried for 15 children with enlarged mediastinal nodes and for children in whom pressure manifestations from enlarged nodes were present. Such therapy relieved the pressure symptoms and consequently made the patient more comfortable. In this way it prolonged life. In some instances in which roentgen therapy was tried it apparently made the condition worse by greatly increasing the already elevated metabolic rate and hastened the fatal outcome. The authors therefore believe that roentgen therapy should be used only for the relief of pressure symptoms or for the relief of localized pain and that such therapy is contraindicated in the absence of these features.—*R. S. Bromer.*

Howes, William E., and Levin, Bella. Lymphosarcoma: a statistical study and evaluation of treatment. *Radiology*, June, 1943, *40*, 565–580.

Lymphosarcoma is highly malignant and though the tumor may disappear under irradiation the general disease progresses. Lymphosarcoma may develop in any organ that has a basic reticulum structure, such as lymphoid tissue, spleen, liver or bone marrow. Illustrative cases in different regions are described and illustrated with roentgenograms, and details of the treatment in the individual cases given.

In general, the authors believe treatment should begin with small doses and gradually increased until a sterilizing dose of 3,000 r has been given to the tumor in three weeks. A diagram is given showing the theoretical location of roentgen portals for treatment of widespread lymphosarcoma. When metastasis has taken place, irradiation should be given not only to the visible tumors but also to the mediastinum and retroperitoneal regions.

From October, 1936, to October, 1940, forty-seven cases of proved lymphosarcoma were admitted to the Brooklyn Cancer Institute. The patients ranged in age from fourteen weeks to seventy-five years. Thirteen of them are still living two to six years after treatment; 9 have no evidence of tumor and appear to have been cured by surgery or irradiation, or both. Among 36 patients with generalized lymphosarcoma, 4 have survived two to six years. In these cases apparently irradiation delayed the course of the generalized disease.

In the discussion Dr. Leucutia suggested that it might be better to give larger doses at one time rather than using the fractional, protracted method advised by Dr. Howes. Dr. Howes said he had sometimes given a large initial dose which was followed by disappearance of the tumor but also by death of the patient.—*Audrey G. Morgan.*

GENERAL

Gage, H. Courtney, and Williams, E. Rohan. The radiological exploration of sinus tracts, fistulae and infected cavities. *Brit. J. Radiol.*, Jan., 1943, *16*, 8–21. Reprinted in *Radiology*, Sept., 1943, *41*, 233–248.

Roentgen examination may show the nature and site of deep-seated sinuses and fistulae which could not be diagnosed in any other way. A high standard of technical precision is necessary in order to explore sinus tracts effectively. The radiologist responsible for the interpretation should perform the actual injection as the pressure variations felt by the injecting hand must be immediately correlated with the visual appearances. The two chief methods of examination are the fluoroscopic control method and the fractional injection method (without pressure release) with roentgenograms taken at suitable intervals. The best contrast medium is iodized oil. Both lipiodol and neohydriol have proved very satisfactory. The technique of the injections and the instruments used are described in detail.

The following classification of sinuses and fistulae based partly on etiological and partly on anatomical grounds is given: (1) sinuses due to infective bone foci; (2) sinuses or fistulae from infected membranous sacs; (3) fistulae having their origin in some visceral infective or ulcerative focus; (4) sinuses or fistulae mainly confined to the superficial fascia; (5) sinuses or fistulae associated with vestigial and developmental anomalies; (6) sinuses or fistulae of simple traumatic origin, by puncture or laceration. Cases of these various types are described and illustrated by roentgenograms. In bone fistulae the contrast between the lipiodol and the bone may not be sufficient for an appreciation of detail in ordinary roentgenograms. In such cases the Potter-Bucky diaphragm and high kilovoltage should be used, even in the limbs. Judgment as to whether this is necessary can be based on the routine preliminary films. In sinuses of infected membranous sacs it is seldom necessary to inject more than 40 cc.; 20 cc. is often

enough. Then changes in the posture of the patient are made until the cavity is completely outlined.

Filling defects in the walls of sinuses are sometimes puzzling. They may be due to the presence of sequestra, to coarse scar contractions, to thick inspissated pus, to calculi or to air bubbles. The globulation of lipiodol, providing continuity of the injection column is maintained, indicates that the oil has entered a fluid medium. If sinuses have not been irrigated recently it suggests the entry of the oil into a pus pocket.—*Audrey G. Morgan.*

Lichstein, Jacob, and Solis-Cohen, Leon. Familial tuberous sclerosis (epiloia) without adenoma sebaceum. *J. Am. M. Ass.*, June 12, 1943, *122*, 429–432.

Although tuberous sclerosis (epiloia, Bourneville's disease) is considered a rare and unusual entity, yet in sixty years from the time Bourneville first described it in 1880 down to 1940, 112 reports have been collected dealing with this disease and its related neurocutaneous syndomes (Recklinghausen's neurofibromatosis, angiomatosis cerebri or Sturge-Weber's disease, and von Hippel-Lindau's disease). In recent years the roentgenologist has become more alert to the significance of metastatic calcifications (calcified plaques) in survey roentgenograms of the skull as an identifying feature of tuberous sclerosis.

The following symptoms may be seen: (1) epileptic seizures, (2) mental deficiency, (3) adenoma sebaceum (acneform eruption, butterfly or bat wing in distribution, involving nasolabial folds and having a trigeminal distribution), (4) congenital tumors of the eye (van der Hoeve's phacoma) and of other organs including rhabdomyomas of the heart and kidney (hypernephroma or Wilms' tumor), (5) periungual fibromas, (6) cystic changes in the small bones, (7) other less frequent congenital defects including such conditions as harelip, cleft palate, pulmonary artery stenosis, microcephaly and accessory digits, and transposition of the great vessels. Sclerotic, potato-like patches are scattered over the surface of the brain, and from this characteristic the name tuberous sclerosis is derived. Subependymal proliferations may be found in the lateral ventricles closely associated with the choroid plexuses. Abortive and latent forms of the disease lacking a fully developed picture have been recognized. Adenoma sebaceum appears to be evidence of a general ectomesodermal disturbance.

The discovery of discrete calcified intracranial deposits renders it incumbent on the observer to incriminate or exclude, in addition to tuberous sclerosis, a number of causes. These may be (1) calcified hemorrhagic foci due to birth injury, (2) tuberculomas, (3) calcified multiple angiomas, (4) parasitic disease (calcified ecchinococcus cysts), (5) disturbance in calcium metabolism as seen in parathyroid disease, (6) old abscesses as a result of meningeal disease or pachymeningitis.

The authors describe 2 cases of familial multiple cerebral calcifications associated with epileptiform seizures. They believe that the additional evidence in 1 case of mental deficiency, congenital eye defect, localization of the calcified masses to the lateral ventricles by pneumoencephalography and the presence of calcifications in the other case in the region of the ventricles justifies the diagnosis of tuberous sclerosis (epiloia). The more frequent use of roentgenography and pneumo-encephalography is recommended in cases of epilepsy to establish the diagnosis of this neurocutaneous syndrome, even in the absence of adenoma sebaceum.—*S. G. Henderson.*

Kerley, Peter. The etiology of erythema nodosum. *Brit. J. Radiol.*, July, 1943, *16*, 199–204.

Most of the studies of the etiology of erythema nodosum have been made on children and young adults. The author has made a careful study of 37 adults aged from eighteen to thirty-three. Generally there was a prodromal stage of three to ten days with malaise and anorexia. This was followed by a temperature of 99 to 101° F., and after twenty-four to forty-eight hours the rash developed. More than half the patients had coryza or pharyngitis with dry cough. Blood counts did not show any consistent abnormality. There were no abnormal physical findings in the chest.

Roentgen examination showed enlarged bronchial glands and pulmonary infiltration in 28 of the cases; in 9 cases there was no evidence of intrathoracic disease. Patients with such findings are frequently diagnosed as tuberculous and sent to sanitaria. These gland enlargements can be differentiated from those of lymphoblastoma by a test dose of roentgen radiation. The glands in erythema nodosum do not respond to such

treatment while those of lymphoblastoma respond quickly. Three types of lung change are described—coarse reticular striation radiating from the hila, widely disseminated, round or oval foci, and a diffuse interstitial fibrosis.

The most common theory of the etiology of erythema nodosum is that it is an allergic reaction to many bacterial and chemical toxins. Other authorities believe that it is a manifestation of tuberculosis. The arguments against the allergic therapy are presented. In all but 1 of these cases all possible drugs and diseases could be eliminated except tuberculosis, Boeck's sarcoidosis and streptococcal infection. The findings in these cases resembled those of sarcoidosis rather than those of tuberculosis. In 2 cases eye lesions strongly suggestive of sarcoidosis dedeloped. The literature on sarcoidosis shows that erythema nodosum develops in about 25 per cent of the cases. The absence of splenic enlargement and phalangeal changes argues against sarcoidosis but all the manifestations of the disease are seldom found in the same individual. The high percentage of cases with swollen and painful joints might be regarded as an argument in favor of rheumatic streptococcal infection but the joint lesions disappeared in a few weeks.

Although no final conclusions can be reached on the basis of 37 cases, the author concludes that at least in the cases with obvious visceral manifestations the condition is a sarcoidosis. Erythema nodosum in adults might be divided into two groups—those with and without visceral manifestations. In the former group the finding of enlarged bronchial glands and lung changes does not indicate tuberculosis.—*Audrey G. Morgan.*

Williams, E. Rohan. Calcinosis. *Brit. J. Radiol.*, Sept., 1943, *16*, 286.

Calcinosis is an interesting form of pathologic calcification. It may occur in a universal or circumscribed form. The circumscribed form generaly occurs in middle-aged or elderly women and is generally confined to the upper limbs, calcium deposits being found in the dermal and subdermal tissues. The fingers are particularly apt to be involved. The universal form generally occurs in children and is much more serious than the form described above. Deposits are found in the skin, subcutaneous, muscular and fascial tissues throughout the body.

Roentgenograms are given of a girl of twelve years of age showing an advanced degree of calcinosis universalis. In the right thigh the adductor group of muscles were involved and in the left the sartorius muscle.

Calcinosis is not a straightforward form of either dystrophic or metastatic calcification but may be a combination of the two types of pathologic calcification.—*Audrey G. Morgan.*

ROENTGEN AND RADIUM THERAPY

Fleming, J. A. C. Carcinoma of the thoracic oesophagus; some notes on its pathology and spread in relation to treatment. *Brit. J. Radiol.*, July, 1943, *16*, 212–216.

The results of treatment of cancer of the esophagus are very poor by any method of treatment. Squamous epithelioma of the esophagus, which is the most frequent type of malignant growth of this region, is radiosensitive and would seem to offer favorable chances for cure. But there is a considerable period between the beginning of the disease and the appearance of symptoms, generally five or six months. Metastases are very apt to occur during this period, making irradiation hopeless. Because of the inaccessibility of the esophagus and its lymphatic field, the presence and extent of metastases cannot be determined by clinical methods. The examination of postmortem material seems to show that there is a definite relationship between the extent of the primary lesion and the degree of metastasis. Generally very long, narrow fields have been used in the roentgen irradiation of cancer of the esophagus but it has been found that if the length of the involved area is more than 5 cm. there is little if any hope of radical cure. If the length of the involved area is less than 5 cm., shorter and wider fields may be used, thus increasing the zone of irradiation around the tumor without increasing the total volume dose given the patient. The width of such fields must be limited, however, sufficiently to avoid injury of the lung fields. Tables are given showing the average length of symptoms before treatment and the average length of survival after treatment.—*Audrey G. Morgan.*

Brown, Samuel, Weiss, H. B., Iglauer, Samuel, and Fine, Archie. Carcinoma of the trachea: report of two cases diagnosed and treated by roentgen rays. *Radiology*, Oct., 1943, *41*, 394–397.

Though carcinoma of the trachea is not so rare as was formerly supposed there has been very little success in its roentgen diagnosis. The authors believe that this failure is due to the use of the ordinary technique and to the practice of stereoscopic examination of the chest in the anteroposterior position. In this position the trachea is practically obscured by the bones of the spine and the sternum. But the oblique or lateral positions show the whole course of the trachea and should always be used with the esophagus filled with barium. The presence and size of the tracheal tumor will be shown by the impression which it makes on the esophagus.

Two cases are described which were diagnosed in this way and given roentgen treatment. The response was good in both cases. One of the patients is alive and in good health after three years and the other died of a complicating heart disease.—*Audrey G. Morgan.*

Fricke, Robert E., and Pastore, Peter N. Radium treatment of granular or hypertrophied lateral pharyngeal tonsillar bands. *Radiology*, Sept., 1943, *41*, 256–260.

The lateral pharyngeal bands are linear patches of lymphoid tissue which lie in the most lateral portion of each side of the pharynx, posterior to the pharyngeal palatine arch, and usually extend into the salpingopharyngeal fold. They tend to hypertrophy after tonsillectomy or adenoidectomy and in that case may cause severe sore throat. In the type of band seen most frequently the surface of the inferior half of the salpingopharyngeal fold has a finely granular appearance. When not acutely inflamed this type of band may readily be overlooked.

As lymphoid tissue is particularly susceptible to irradiation, this method of treatment has been successfully used in these cases. The authors discuss 24 cases treated at the Mayo Clinic in all of which the results were good, sore throat and frequent colds being overcome and the general health improved. They have devised a simple and practical applicator for these cases, which is illustrated. A metal rod is threaded at the end so that a brass and silver tube containing radon can be attached; there is a hinged joint just behind the attachment. The end of the rod with the tube containing the radon attached is passed through the nostril; on turning, the hinged portion drops downward and lies against the hypertrophied band with the long axis of the radon pressing against the lateral pharyngeal wall.

The effect of irradiation on lymphoid tissue is transient and it may be necessary to repeat the treatment in some of these cases.—*Audrey G. Morgan.*

Mandeville, F. B. Roentgen therapy of orbital-pituitary portals for progressive exophthalmos following subtotal thyroidectomy. *Radiology*, Sept., 1943, *41*, 268–271.

Malignant or progressive exophthalmos is a form of the disease which tends to progress after subtotal thyroidectomy. Since 1929 varying opinions have been expressed as to the value of treating such cases by irradiation of the orbits. The literature of the subject is reviewed and one case is described which was treated by the author with good results.

The patient was a trained nurse fifty years of age on whom thyroidectomy was performed for exophthalmic goiter. About one year after the operation progressive exophthalmos was noticed which grew worse under the treatment of general physicians and ophthalmologists. Roentgen treatment was started nine months after this exophthalmos began and twenty-one months after the subtotal thyroidectomy. Treatment was given to both right and left temporal regions, using 200 kv., on a Villard circuit with tube in air, half-value layer 1.8 mm. copper, filter 2 mm. copper and 1 mm. aluminum, 20 ma., 50 cm. distance and fields 6×8 cm., angling posterior to the eyeball and toward the pituitary. Each side was given a dose of 150 r on alternate days, up to a total of 900 r on each side, well over 1,000 r on the skin. She had discomfort consisting of swelling of the orbital tissues and lacrimation for about two weeks, after which the exophthalmos receded; there was remarkable improvement in vision and movement of the eyeballs was no longer limited. Pain disappeared. Eight weeks later, a second series was given with lower voltage and less filtration with a total dose of 600 r in air on each side. There was a mild reaction two weeks after the second series which lasted only a few days. Four months after treatment the patient was clinically well and carrying on her daily duties.—*Audrey G. Morgan.*

Leddy, Eugene T. Roentgen therapy for bronchiogenic carcinoma. *Radiology*, Sept., 1943, *41*, 249–255.

Surgery is still to be recommended for operable cases of bronchiogenic carcinoma, but the author finds irradiation an excellent palliative measure in inoperable cases or those in which the patient refuses operation. Reports in regard to treatment and results are quoted from various authors. Some have claimed that no prolongation of life results from roentgen treatment but this is disproved by a series of 250 cases treated by the author and Moersch. Roentgen therapy was used in 125 cases and in the other 125 cases which were used as controls neither roentgen therapy nor surgery was used. All of the cases in which roentgen therapy was used were inoperable. In the group of cases in which no treatment was given none of the patients lived more than a year after the diagnosis was made. But 25 of the 125 cases in which roentgen treatment was used lived from one to twelve years. Five of the patients lived more than five years.

In most cases of malignant disease of the lungs the patients are debilitated and past middle age and are therefore poor subjects for any form of treatment. As palliative results are the most that can be expected in most cases, careful clinical judgment must be used in regard to the method of treatment. The author has found that the simple cross-firing of the lesion with moderate doses of roentgen rays has been as valuable clinically as that obtained by more radical methods and at the Mayo Clinic, where this work was done, the results have not been in proportion to the size of the dose. The technique used there is described in detail. As the injuries reported by some authors were due to the large doses the way to prevent them seems obvious.—*Audrey G. Morgan.*

DANN, DAVID S., and KORITSCHONER, ROBERT. Preoperative roentgen therapy of breast carcinoma: analysis of histologic reaction and roentgen technic. *Radiology*, Sept., 1943, *41*, 213–224.

The authors discuss 10 cases given preoperative roentgen treatment. Detailed histories and pathologic reports are given and the pathologic findings illustrated by photomicrographs. Two techniques were used: the first used in all but one of the cases was: 200 kv.; 20 ma.; 1 mm. copper and 1 mm. aluminum filter, 80 cm. distance, half-value layer 1.425 mm. copper; 9.16 r (in air) per minute. The daily dose to the skin was 100 r (in air); the time of administration was 10.91 minutes. Two breast ports and, after completion of the breast irradiation, three axillary ports were treated daily with cones. The apparatus used was a General Electric KX-3, half-wave rectification. The technique used in the other case was: 250 kv., 15 ma.; Thoraeus filter plus 1 mm. aluminum; 50 cm. distance; half-value layer 2.80 mm. copper; 33.22 r (in air) per minute. The daily dose to the skin was 100 r (in air) in 3.6 minutes. Three cycles of treatment were given, each of fifteen days, with an interval of thirty-five days between the first and second and of forty-three days between the second and third. The total estimated breast tumor dose was 1,876 r in each cycle, the mid-axillary dose 1,513 r in each cycle. Two breast ports and two axillary ports were treated daily without cones. The apparatus was a General Electric 250, self-rectification.

The histological findings are discussed and special attention called to "spotty necrosis." This means that certain tissue particles may not be affected by irradiation at all. This "target hypothesis" may explain the failure of irradiation to bring about complete sterilization of these tumors. Preoperative irradiation of carcinoma of the breast does accomplish definite reduction of the size of the tumor and regressive changes which may be due to the fact that the host's defensive reaction is increased. Inoperable carcinoma may become operable after irradiation. Irradiation is recommended for inoperable cases, for operable cases in which operation is refused and for operable cases in which there are physical conditions which contraindicate operation. Further studies of the individual factors governing irradiation may lead to an improved technique for the irradiation of carcinoma of the breast.

In order to carry out such studies effectively the authors recommend the establishment of a Commission of representative radiologists, surgeons and pathologists to be appointed to plan and carry out a study of this problem. As judgment must be based on a large material, various institutions should be selected and allotted special points in the program for study. Each institution should submit to the Commission a detailed report of each individual case when the treatment is completed and follow-up observations should be made at regular intervals. Based on such detailed information in large numbers of cases the Commission can decide on the value of preoperative irradiation in general

and the comparative merits of the different methods.—*Audrey G. Morgan.*

Brewer, Arthur A., and Zink, Oscar C. Radiation therapy of acute subdeltoid bursitis. *J. Am. M. Ass.*, July 17, 1943, *122*, 800.

This condition has been designated by a variety of names: subdeltoid bursitis, calcarea peritendinitis, periarthritis of the shoulder, calcifications of the supraspinatus tendon or simply painful shoulder. By common usage it is most often called subdeltoid bursitis. Acute subdeltoid bursitis presents a very typical clinical picture. There is a rather sudden onset of severe pain in the shoulder, usually with no history of significant trauma. Exquisite point tenderness may be elicited over the area of the subdeltoid bursa, with pain radiating down the arm, limitation of motion, and muscle spasm. In typical cases the diagnosis of bursitis frequently will be verified by the demonstration of a deposit of calcium just lateral to the greater tubercle of the humerus. Chronic cases present similar but much less pronounced symptoms. The duration is several weeks, months or longer. The onset is usually insidious, although an occasional acute case may become chronic because of inadequate treatment. There is usually decided limitation of motion.

The treatment of choice should be one offering the shortest period of disability. With this in mind the authors have reviewed a series of cases treated with radiation. The physical factors employed are 200 kilovolts (constant potential), 18 milliamperes, 0.5 mm. of copper plus 1.0 mm. of aluminum filter (1.18 mm. of copper half-value layer), 50 cm. target-skin distance and a 10 by 15 cm. port directed toward the anterolateral aspect of the involved shoulder. A single treatment with a dose of 300 roentgens is usually sufficient, although this may be repeated in from seven to ten days if there has been definite but incomplete relief. The typical response in an acute case is a frequent but not inevitable aggravation of symptoms for eight to twenty-four hours. This short period of aggravation is followed by rapid relief of pain and limitation of motion, so that in most instances the patient is able to resume his normal activities in from twenty-four to forty-eight hours after treatment. The calcium deposits will be absent in many instances if roentgenograms are repeated in a few weeks. If there has been no improvement at the end of forty-eight hours operative treatment should be considered.

Approximately 30 per cent of the patients with chronic bursitis are improved in varying degrees, but only an occasional patient is cured by this method. If definite symptomatic relief is not apparent within ten days after treatment, the treatment is considered a failure. However, these shoulders respond poorly to any type of therapy, and it seems reasonable to submit such a patient to a trial of radiation therapy before instituting more radical measures.—*S. G. Henderson.*

Barden, Stuart P. Healing of radiation fractures of the necks of the femora, with a report of a case. *Radiology*, Oct., 1943, *41*, 389–394.

Post-irradiation fractures of the necks of the femurs rarely heal or do so only by fibrous union. The bone changes in these fractures seem to be directly proportional to the amount of radiation absorbed. A case of fracture of the necks of both femurs with complete bony healing is described and illustrated with roentgenograms. The patient was a woman of sixty-five who was being treated for a basal cell epithelioma of the urethra. She began to complain of pain in the left hip and examination showed fracture of the neck of the femur. Two years later the roentgen evidence of fracture had disappeared. Two and a half years after the fracture of the left femur the process was repeated in the right hip. This fracture also healed with bony union. No treatment was given except rest in bed and limited traction. There seemed to be no reason for the difference in time of the fractures except that a greater number of roentgens was delivered to the left femur.

As most of these fractures occur in the treatment of cancer of the cervix, the author uses small portals in this treatment, not larger than 12 cm. in diameter. When using the posterior portal the patient is cautioned to turn her toes inward and her heels outward, thereby rotating the femurs internally so that the necks of the femurs are farther away from the source of radiation.—*Audrey G. Morgan.*